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ORIGINAL LECTURES.

THE CARTWRIGHT LECTURES. ON THE GENERAL PATHOLOGY OF FEVER.

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LECTURE I.

THE NATURE OF FEVER.

(Concluded from page 371)

THAT the heat-regulating mechanism, although profoundly disturbed, is not entirely paralyzed in fever, is proven by the effect of heat and cold upon fevered individuals. Although Colasanti believed that his fevered guinea-pig had lost all power of heat regulation under varying external temperatures, a similar conclusion has not been reached by others who have investigated this question.

We can best describe the condition of heat regulation in fever as unstable or ataxic. External cold stimulates to some extent the heat production of an individual in fever, but by no means as much as it does in health. A person in fever is not able to maintain his bodily temperature under the influence of heat and cold to the same degree that he can under normal conditions. Liebermeister, as is well known, held that heat regulation in fever is simply adjusted for a higher point, although he admits not perfectly adjusted. Experience shows that this so-called adjustment is so unstable that it does not seem proper to compare it with that to normal temperatures in health, so that it is not clear what deep meaning lies in Liebermeister's idea.

We cannot imagine the heat-regulating mechanism to be other than a nervous one.

Some years ago this was about as far as the theory of the mechanism of fever could be carried. All paths led to this mysterious nervous apparatus, and beyond this nearly all was speculation. We stood, as has been said, before imposing processes veiled in the deepest obscurity. Since then the veil has been lifted here and there and we have caught glimpses of the nature of these processes. I refer particularly to the results of researches which have brought to light a more immediate and direct dependence upon nerve energy, than had been supposed, of chemical processes concerned in the disintegration and construction of tissue, and, therefore, in the production of heat.

With one side of the nervous mechanism concerned in temperature regulation, the classical investigations of Claude Bernard have made us tolerably familiar. This is the vaso-motor nervous apparatus presiding over the circulation of blood in the superficial parts of the body, and thereby controlling in great measure the discharge of heat. That the important part taken by the perspiration in the dissipation of heat is likewise under nervous influence, has been demonstrated by Luchsinger. The facts concerning this side of the regulation of heat are too well known to require elucidation on this occasion.

Heat regulation, however, is effected not only by variations in the elimination of heat, but also by changes in the production of heat. Familiar as the fact is, it can never cease to arouse our admiration that the temperature of the body remains the same in cold and in warm atmospheres. Man has become so dependent upon clothing that in the naked condition his capacity of preserving his normal temperature in a cold environment is much less than that of most animals. Pflüger has demonstrated that the heat regulation under varying external temperatures is accomplished by changes both in heat production and in heat loss, so that in a cold atmosphere more heat is produced and in a warm atmosphere less heat, provided the external temperature is not so high or so low as to make it physically impossible to preserve the body temperature. It is evident that this is the most rational and economical method of retaining the internal temperature of the body. To regulate the body temperature simply by variations in the discharge of heat, as was formerly supposed to be the method, would be, as has been said, like regulating the temperature of our rooms summer and winter by opening and shutting the windows without controlling the source of heat.

A heat-producing or thermogenic apparatus, therefore, is no less a part of the heat-regulating mechanism than is the heat-discharging or thermolytic apparatus, to use the terms employed by Foster. As the thermogenic apparatus is less generally understood it is not permissible to dismiss its physiology in this connection so briefly as I have the thermolytic, although our knowledge of the former is very imperfect.

I can assume that the convincing reasons are known to you which have led physiologists to conclude that most of the animal heat is produced in the muscles and the glands, and that the muscles have the larger share in this function. It is also well known that stimulation of secretory and motor nerves causes not only visible physical alterations in the glands and muscles, but also production of heat. This sort of dependence of heat production upon innervation has been long admitted. It may, however, not be so generally known that there are reasons to believe that nervous impulses control chemical changes which result in the production of heat independently of visible physical alterations of the tissues; in other words, that heat production or thermogenesis is at least in considerable part under the immediate and direct control of the nervous system. The idea is not a new one, and was advocated especially by Claude Bernard. Recent discoveries, however, have given it unexpected support.

This subject of the relation of innervation to thermo-

genesis is most pertinent to the pathology of fever, but it is essentially a physiological one, and as I wish to confine to a single lecture what I have to say concerning the theory of fever, it is impossible for me to do more than summarize the most essential points belonging here. This I can do the more readily as Dr. MacAlister,[1] in his admirable Goulstonian Lectures on the "Nature of Fever," which were delivered last year, has clearly and forcibly presented the main facts.

The larger part of these facts relate to the chemical changes and heat production of muscles under varying conditions.

That a large part of the chemical changes in a muscle in the condition which we call repose is under the influence of the nervous system, is made apparent by the great diminution in its consumption of oxygen and formation of carbonic acid, which follows the separation of the muscle from all connection with the central nervous system. This is conclusively shown by the experiments of Bernard, Pflüger, von Frey, and others. It is, of course, possible, and it has generally been supposed that under these circumstances it is simply the withdrawal of motor impulses which lowers the heat-producing energy of the muscle.

Far more suggestive as regards the point under consideration are the results of investigations which have been carried on in Ludwig's laboratory by Meade Smith, MacAlister, and Lukjanow. These experimenters have shown that heat production and contraction are in a measure independent properties of the muscle. By various influences the thermogenic property may be so impaired that a stimulus causes contraction with scarcely any development of heat. The laws governing the restoration and the fatigue of the thermogenic function differ from those controlling the mechanical function. These researches, upon which I here only touch, have made it extremely probable that there are in the muscle chemical processes resulting mainly in the production of heat and chemical processes causing mainly contraction, and that these processes, although coördinate, are not identical. A new light is shed upon the meaning of the term chemical tonus of muscle, which has for some time been used by some physiologists. Great caution is properly exercised by Ludwig and his pupils in the interpretation of these interesting results. They do not infer from them, necessarily, the existence of so-called thermic or calorific nerves. They suggest that it is possible to explain the phenomena upon the supposition that there are in the muscle two kinds of material, thermogenic and contractile, and the nervous impulses acting upon these may pass through the same set of nerves.

Proof of the existence in connection with muscles of thermic in distinction from motor nerves, would be afforded if we could succeed, after paralysis of the motor nerves, in exciting by nerve stimulation the thermogenic function of the muscle. We possess, in curare, a drug which paralyzes the terminations of the motor nerves. Meade Smith attempted by its aid to determine whether possible thermic nerves may be differentiated from motor, but he reached no positive conclusion on this point. There are, however, on record some observations which suggest the possibility that in moderate doses curare may leave intact thermic nerves after the suspension of the function of motor nerves. When an animal is profoundly under the influence of curare the internal temperature falls, and the processes of oxidation are greatly reduced. The animal is no longer able to resist the changes of external temperature, its own temperature rising and falling like that of a cold-blooded animal when exposed to heat or cold. This effect of curare poisoning is another proof of the dependence of heat production upon nervous influences. Claude Bernard in his early researches on the action of curare noted elevation of temperature soon after its administration. Voison and Lionville[1] observed after subcutaneous injections of curare in man rigors, perspiration, headache, and elevation of temperature to 104.7° F. They attribute to curare the power of producing all of the essential phenomena of fever. Fleischer[2] noticed in dogs and in rabbits rise of temperature after curare injections, and the same was observed in a rabbit in one case by Högyes.[3] Recently Mosso[4] claims that an animal may be placed so far under the influence of curare as to paralyze completely the voluntary muscles, and the internal temperature either remain normal or rise. He infers that these moderate doses of curare, although sufficient to paralyze the motor nerves, must have left intact the thermic nerves. That rise of internal temperature is not due to retention of heat, he thinks is evident from the normal or elevated temperature of the skin. Mosso is strengthened still further in his conclusion as to the existence of thermic nerves, by finding that after complete paralysis of the motor nerves by curare injections of strychnine cause an elevation of rectal temperature, which may amount to three degrees centigrade. Under these circumstances strychnine produces no spasms or other visible mechanical effect upon the muscles. Mosso brings forward a number of other experiments (decidedly open to criticism) intended to demonstrate the existence of nerves directly controlling heat production, but those which I have mentioned are by far the most striking. It seems to me that the natural interpretation of these experiments is in favor of the view that there are nerves controlling heat production in the muscle distinct from motor nerves. Far more conclusive as to this point than thermometric observations would be calorimetric experiments determining the heat production of animals under varying doses of curare.

This whole line of experimentation directed toward the differentiation of the mechanical and the chemical functions of muscle is certainly most suggestive, but so long as the interpretation of the results is not perfectly clear, we should be very guarded in drawing far-reaching conclusions. I cannot refrain, however, from pointing out that, as mentioned by MacAlister, all of these thermogenic phenomena may be found eventually to depend upon nerves whose chief function, on the one hand, is the disintegration, the metabolism of tissue, and, on the other hand, the restoration, the anabolism of tissue. From the study of the electrical changes which stimulation of the pneumogastric nerve produces in the heart muscle, Gaskell concludes that this nerve puts the heart in a condition of relative rest during which the energy of the muscle is increased. During this period there is reason to believe

[1] MacAlister: The Lancet, 1887, vol 1.

[1] Virchow and Lionville: Virchow u. Hirsch's "Jahresbericht," 1886, I. p. 330.

[2] Fleischer: Pflüger's Archiv, Bd. ii. p. 441.

[3] Högyes: Arch. f. exp. Path. u. Pharm., Bd. xiv. p. 136.

[4] Mosso: Virchow's Archiv, Bd. 106.

that the material of the muscle, which, when disintegrated, gives rise to heat and mechanical work, is in the process of restoration. Hence Gaskell speaks of the pneumogastric or inhibitory nerve of the heart as anabolic—that is, it directs the restorative, formative, anabolic processes in the muscle. On the other hand, the accelerator nerve of the heart induces the opposite electrical changes in the heart muscle. Gaskell describes this nerve as katabolic—that is, its stimulation causes disintegration of the muscle materials, and liberates energy in the form of heat and of mechanical work. If it be found that similar inhibitory and accelerator nerves preside over the chemical changes in the voluntary muscles and other tissues of the body, then Gaskell's induction as to the existence of anabolic and katabolic nerves must be regarded as one of the most important and profound in modern physiology. Thermo-excitory nerves we should then rank as katabolic, thermo-inhibitory as anabolic.

These investigations tending to demonstrate the independent existence of thermogenic properties in the muscles and possibly of nerves directly controlling thermogenesis, have prepared us for the consideration of the relation of the central nervous system to the heat-producing properties of the body. Here you will willingly permit me to confine my remarks to the more essential and best established facts, without entering into a full discussion of one of the most perplexing subjects in the physiology of the nervous system.

The clinical basis of the doctrine that lesions of the central nervous system influence directly the temperature of the body was laid by Sir Benjamin Brodie, who reported the well-known case of fracture of the cervical vertebræ and injury of the spinal cord followed within a few hours by a rise of temperature to 111° F. measured between the scrotum and the thigh. Since then many similar instances have been reported. It is well to remember, in framing theories on the basis of these cases, that there are also on record not a few instances in which apparently similar injuries of the same parts of the spinal cord have been followed by equally striking fall of temperature.

The experimental basis for the acceptance of an influence of the nervous system on temperature was laid by Bernard, in his celebrated experiments on the effects of division of the sympathetic nerve in the neck. Bernard interpreted the increased temperature of the ear following this operation as referable not only to the larger amount of blood in the part, but also to increased tissue metamorphosis and consequent heightened heat production. The latter part of this interpretation is not generally accepted. It may be said here that the common idea that an organ or tissue simply because it receives a larger supply of blood indulges in more active metabolism is opposed by the investigations of Pflüger, who finds that the amount of oxygen taken up by the cells depends in a far higher degree upon the state of their innervation at the time than it does upon the supply of oxygen. The demand of the tissues for oxygen is not increased simply because the supply is greater. The arguments upon which Pflüger bases this line of reasoning, although not without opposition, would dispose of the idea that when any considerable increase of heat production in the muscles occurs this can be explained simply by vasomotor changes. I mention this here, because the opinion has been advanced that a large part of the increased production of heat in fever and after certain injuries to the nervous system is referable simply to vasomotor changes in the muscles.

In 1866 Tscheschichin[1] published experiments which he interpreted as indicating the existence in the brain of centres which, when irritated, moderate the production of heat, and which he called heat-moderating or heat-inhibitory centres, and in the spinal cord of centres which when stimulated excite the production of heat, heat-excitive centres. These conclusions were not justified by the experiments, but they have been widely accepted.

I may here say that, in this connection, I use the term heat centre as a convenient and generally adopted one. There is a proper reaction against the prevailing misuse of the word centre for all sorts of little understood localization of nervous functions. It would undoubtedly be more accurate to use some such expression as thermically active region instead of heat centre.

It is not easy to reconcile the clinical fact that in human beings lesions of the spinal cord may be followed at once, or in a very short time, by extraordinary elevations of temperature with the results of experiments on animals. That the rise of temperature in human beings is not due to inflammatory fever is apparent from the rapidity with which it follows the injury. It cannot be explained by vasomotor lesions, for the paralysis of the vasomotor nerves accelerates the discharge of heat from the surface of the body. The high temperature can be explained on Tscheschichin's assumption. The lesion either stimulates the spinal thermogenic centres or removes the influence of the thermo-inhibitory centres. These clinical observations are the strongest support which has been found for the belief in the existence of centres in the spinal cord which accelerate heat production. Complete section of the cervical part of the spinal cord in a dog or a rabbit is, under ordinary conditions, always followed by a rapid fall of the internal temperature and diminished oxidation. These varying results in man and in animals have been explained by supposing that after section of the cervical cord, on the one hand, the discharge of heat is increased by dilatation of the superficial bloodvessels, in consequence of vasomotor paralysis; and, on the other hand, the production of heat is increased by withdrawal of thermo-inhibitory cerebral impulses. If, as ordinarily happens in dogs and smaller animals, the first factor predominates, then, in consequence of sinking of the internal temperature, the heat-producing processes are so reduced that the influence of the second factor is not manifest. The attempt has been made to test this explanation by placing the animal in a warm atmosphere. If thereby the heat discharge be reduced to a minimum, it is found that the internal temperature of the animal often rises more rapidly than that of a normal one under the same external conditions. Here thermometric observations are not conclusive as to the point to be tested, for the more rapid rise can be explained simply by failure of the heat-regulating mechanism. Wood's calorimetric experiments seem to show that there is greater heat production, under these circumstances, in the animal with cut cord. These experiments admit of various interpretations, but if they be regarded as establishing the assumption from which we started, then it is

[1] Tscheschichin: Reichert und Du Bois-Reymond's Archiv, 1866.

evident that in man and in large animals the increased heat production after injury of the cord would not be so readily overcome by the increased discharge of heat from the surface, for, in proportion to its volume, a large animal has less surface than a small one. The interplay, therefore, of these opposing tendencies might cause different results, according to the size of the animals.

Tscheschichin found that a transverse section made at the junction of the pons and medulla oblongata is followed, in a short time, by rise of internal temperature. As the superficial temperature is also elevated, he concludes that there is no retention of heat; moreover, the section is made above the dominant vasomotor centre. Wood has shown by means of the calorimeter that after this operation there is actual increase in the production of heat. He, as well as Tscheschichin, interprets the experiment as indicating thermo-inhibitory centres above the lower border of the pons. Bruck and Günter[1] repeated and modified these experiments under Heidenhain's directions. Out of seven cases in which they separated the pons from the medulla, they observed in only two rise of temperature; of eleven cases in which they punctured the pons with a needle, in five they noted increased temperature. Heidenhain thinks these experiments indicate heat-exciting rather than heat-moderating centres, and it must be admitted that the evidence is not conclusive in support of either view.

By far the most interesting and conclusive experiments, showing the influence of the central nervous system on thermogenesis are those of Isaac Ott, followed by Richet, Aronsohn and Sachs, Baginsky, and Girard.[2] The observations of Ott, Richet, and Aronsohn and Sachs were made independently and at about the same time, but Ott was the pioneer. The experiments of Aronsohn and Sachs are reported with especial fulness and detail. These investigators found that if the skull of a rabbit be trephined at the junction of the coronal and sagittal suture, and a needle be passed vertically down so as to puncture the anterior part of the caudate nucleus near its median convexity, there follows a rise of temperature, which may amount to three degrees or more, and which may persist for two or three days. The rapidity and the duration of this elevation of temperature vary somewhat with the depth of the puncture, parts immediately beneath the caudate nucleus being also thermically active. Puncture of the overlying cortex or medullary substance has no such effect upon temperature. With the exception of some increase in the frequency of the respiration and of the pulse, the animal after puncture of the anterior median part of the caudate nucleus presents no abnormal symptom other than the rise of temperature.

I have several times repeated this experiment and always with the result described. That the pyrexia induced by puncture of the caudate nucleus is not due to vasomotor changes causing retention of heat, is proven by Richet's and Ott's calorimetric experiments, and by the determination by Aronsohn and Sachs that the consumption of oxygen and the elimination of carbonic acid, and urinary nitrogen are increased. Aronsohn and Sachs and Girard find that electrical stimulation of the anterior median part of the caudate nucleus causes the same thermic phenomena as the puncture, and they, therefore, conclude that this region contains a thermo-excitor heat centre. This is the natural interpretation of their experiment, although Baginsky and Ott regard the centre as thermo-inhibitory on grounds which cannot be considered convincing.

Ott claims that there are four cerebral heat centres, one about the corpus striatum, the second in the caudate nucleus, the third in the anterior inner end of the optic thalamus, and the fourth near the median line between the optic thalamus and the corpus striatum. The greatest rise of temperature he found after injury of the thalamic centre.

No adequate demonstration has been afforded of any influence of the cerebral hemispheres upon thermogenesis. Corin and van Beneden[1] find that pigeons, after removal of their cerebral hemispheres, exhibit no change of temperature, no failure of the heat-regulating mechanism, and no alteration in the excretion of carbonic acid.

I have endeavored to present to you the main physiological facts bearing upon the relation of the nervous system to thermogenesis. It must be admitted that we are left to surmise as to the interpretations of many of the facts, and particularly as to their connection with each other. But certain important points come out clearly. We have found evidence of the existence in the body of chemical processes resulting chiefly in the production of heat energy. We have learned that these processes are under the direct control of the nervous system, and possibly of nerves distinct from those now recognized as motor or secretory. We have seen that there are regions in the central nervous system which are doubtless in some way connected with these nerves, and through them control the chemical processes resulting in the production of heat.

The bearing of these facts upon the theory of fever is evident. The study of heat production and of heat loss in fever has led us, by arguments which need not be repeated, to the conclusion that the pyrogenic agent must in some way act upon the heat-regulating mechanism. The study of this mechanism, more particularly of its thermogenic side, affords some insight into the manner in which the fever-producing agent may affect the regulation of heat. The main difficulty in the neurotic theory of fever has been to understand how by any action of the fever-producing agent directly upon the nervous system the chemical processes leading to heat production could be stimulated. I have dwelt thus at length upon the innervation of thermogenesis to show that this difficulty has been in great part overcome.

To some it seems more reasonable to suppose that the pyrogenic agent circulating in the blood acts directly upon the tissues, altering and stimulating their chemical changes. This is the hæmic theory, which, in some form, has always stood over against the neurotic theory of fever. Although it may at first glance appear simpler, the hæmic theory is really the more complicated, for it has already been set forth that we cannot explain fever simply by increased heat production, so that even if the primary effect of the fever agent were upon the heat-pro-

[1] Bruck and Günter: Pflüger's Archiv, Bd. iii.

[2] Ott: Journal of Nervous and Mental Diseases, April, 1884; MEDICAL NEWS, July, 1885. Therapeutic Gazette, Sept. 1887. Richet: Arch. de Phys., 1884. Aronsohn and Sachs: Pflüger's Archiv, Bd. 37. Baginsky and Lehman, Virchow's Archiv, Bd. 106. Girard: Arch. de Phys., 1886.

[1] Corin and van Beneden: Arch. de Biol., 1887.

ducing processes there must be a secondary influence upon the nervous system, for heat dissipation is no less disturbed than heat production.

If an animal be thoroughly curarized so that no impulses from the nervous centres can reach the muscles, the great heat producers, then it is found to be impossible to produce febrile elevation of temperature by the injection of pyrogenic agents. This fact, first demonstrated by Zuntz, is justly held to weigh heavily in favor of the neurotic theory of fever.

Contrary to the results of Murri,[1] I have not been able to induce rise of temperature or check its fall by the injection of pyrogenic agents into the jugular veins of dogs whose spinal cords have been cut in the lower cervical region. In these experiments I have employed various pyrogenic agents, and especially pepsin and papoid. One specimen of the latter which I used possessed very striking pyrogenic properties.

The pyrexia produced by puncture of the caudate nucleus in the manner already described, possesses all of the essential properties of fever, regarded as abnormal elevation of temperature. In this experimental condition there are increased production of heat, increased dissipation of heat, excessive elimination of urea and of carbonic acid, and excessive absorption of oxygen. The breathing and the pulse are increased in frequency. The elevation of temperature (usually after a brief fall) begins within half an hour after the puncture, attains a febrile height and persists sometimes for days. As no observations exist as to the heat regulation of these animals, I have made experiments on rabbits after puncture of the caudate nucleus, by placing them in a cold environment and in a box heated to various temperatures, and I find that their power of temperature regulation is less than that of normal animals.

These animals, in a word, present all of the essential symptoms of fever, and I do not know why we should not call the condition fever. If so, we must admit that injury to a circumscribed definite region of the brain is capable of causing fever. This experiment, therefore, is of the greatest importance in support of the neurotic doctrine of fever. It indicates, of course, that we may have fever of purely nervous orgin, without any pyrogenic agent in the blood. This experimental evidence is supported by the clinical cases collected by White in *Guy's Hospital Reports*, 1884. It is to be hoped that by careful thermometric study of focal brain and cord lesions a more accurate idea may be reached than is now possible of the topography of the thermically active regions in the central nervous system of man.

Admitting the dependence of fever upon the nervous system, I do not regard as particularly profitable with our present knowledge the discussion as to whether febrile thermogenesis is excited by the withdrawal of thermo-inhibitory impulses or by the stimulation of thermo-excitory nerves or nerve centres. If we regard, and there are forcible arguments for doing so, possible heat-inhibitory nerves as anabolic, and the heat-exciting nerves as katabolic, then inasmuch as the formative or anabolic processes are manifestly in abeyance in fever, we can reasonably infer that the function of the heat inhibitory nerves or centres is impaired. The phenomena of the febrile chill in which both the contractile and the thermogenic properties of muscle are stimulated, speak strongly in favor of direct irritation of the heat-exciting nerves in fever. We might infer, therefore, that both sets of nerves or nerve centres are affected.

But my aim in this lecture has been not so much to construct a theory of fever, a theory which, although it may be useful, must necessarily be largely speculative if it be coherent and rounded, but to bring before you the main facts concerning heat production, heat loss, and heat regulation in fever, and to point out the physiological basis on which their solution is to be expected.

[1] Murri: Teoria della Febbra, Fermo, 1874.

LECTURE II.

Delivered April 5th.

THE EFFECTS OF INCREASED TEMPERATURE OF THE BODY.

IN the last lecture I endeavored to bring before you the main facts which we possess bearing upon the explanation of febrile rise of temperature. After a review of our knowledge concerning heat production, heat dissipation, and heat regulation in fever, we were led to the conclusion that neither the changes hitherto observed in the production of heat nor those in the loss of heat suffice to explain febrile temperatures. Such an explanation seemed possible only upon the assumption that the fever-producing agents act either directly or indirectly upon the nervous mechanism controlling the relations to each other of the production and the discharge of heat. We then turned our attention to the relations of the nervous system to these processes, and found that the investigations of recent years have enabled us to obtain a much clearer conception than was formerly possible, of the manner in which the complicated heat relations in fever may be brought about by an action upon the nervous system. We found even substantial experimental basis in support of clinical facts which show that circumscribed lesions of certain parts of the central nervous system may induce directly febrile disturbances of animal heat.

To carry the subject further, to consider whether pyrogenic agents act primarily or only secondarily upon the processes concerned in heat production or upon those concerned in heat dissipation, or upon the apparatus regulating the relations to each other of these two sets of processes, to discuss whether these agents act directly upon the central nervous system, and if so upon what part; or upon the peripheral nerves or their terminations, or upon the tissues; to follow out more fully than we have done hitherto the themes here suggested would lead us with our present knowledge into a sea of speculation. We should find only here and there a faint light of fact to guide us. Even upon foundations as insecure as this speculations have their legitimate uses. A good hypothesis is a most valuable incentive to scientific work. It is not my purpose, however, to carry you with me further in this direction, although I realize that a single lecture has been far from sufficient for a thorough exposition of this subject.

After dismissing the considerations bearing upon the so-called theory of fever, there still remains a host of questions properly belonging to our subject. It is evidently impossible, even if it were desirable, that these lectures should include a discussion of all of these questions. I have, therefore, selected certain ones, partly be-

cause they seem to me of immediate interest and partly because I have given some attention to their study.

In the present lecture I wish to present to you some observations concerning the relation of elevation of temperature to other disorders of fever, and as to the question how far increased temperature is a source of danger in fever?

Here I may repeat that the word fever is used as a convenient name for a group of symptoms commonly associated together in febrile diseases. The association of these symptoms, however, is so loose that we regard only the dominant one—the increased temperature—as the essential criterion of the existence of fever. It saves circumlocution to adopt this somewhat vague and common signification of the term fever, although I think it would be an improvement to confine the term to abnormal elevation of temperature. The literature of fever is full of misapprehensions resulting from the various meanings attached to the word by different authors.

All of the bodily functions may be disordered in fevers. The various symptoms or classes of symptoms which are so commonly associated as to be regarded by many as belonging to the febrile process are, in addition to heightened temperature, increased frequency of the pulse and other circulatory disturbances, increased rapidity of respiration, muscular weakness, lessened secretions, disordered nutrition and digestion, and nervous symptoms.

What is the connection, if any, between these symptoms and the elevation of temperature? What degrees of elevated temperature are dangerous to life, and in what does the danger consist? This subject can, at least, claim the interest that attaches to the questions of the day. The various opinions which have been held by clinicians on these points are too well known to you to require an historical review. I need only remind you that until within a few years the views advocated with especial force for nearly thirty years by Liebermeister have prevailed, although not without considerable opposition. According to these views, the chief source of danger in uncomplicated essential fevers is the elevation of temperature, and the main indication for treatment is the reduction of temperature. Above all, it was urged with apparently convincing arguments that the weakness of the heart, which is undoubtedly one of the gravest dangers of fevers, is the direct effect of prolonged high temperature, and is manifested anatomically by parenchymatous or fatty degeneration of the cardiac muscle. Liebermeister sharply defined his position when he said, "A man whose temperature measures continuously 104° (40° C.) or more surely dies in consequence of the elevation of temperature, one in a few days, another after a somewhat longer time, according to the resistance of the individual." "If his temperature reaches 108.5° (42.5° C.) or more, then is he irrecoverably lost."[1]

At the present moment there is a decided reaction against these views, a reaction which in some quarters goes to the extent not only of denying that there is danger in febrile temperatures which do not exceed a very high point, but of asserting that the elevation of temperature is a beneficent provision, a most important *vis medicatrix naturæ*, which should not be checked by the interference of the physician. This reaction of opinion is plainly due, in great part, to the disappointment which has followed the high hopes raised by the discovery of a number of drugs which are admirable antithermic agents and, nevertheless, do not exert over febrile diseases that controlling influence which had been anticipated.

These questions, you may say, are clinical ones, and not much edification is to be expected from their discussion by a pathologist. So far as the propriety of the use of antipyretic agents or of any other mode of treatment in fever is concerned, it is true that the decision must be reached at the bedside and cannot and never should be controlled by the results of experimental pathology. But an appeal has properly been made to experimental pathology to shed light upon such questions as the effects of heat upon the functions of the whole body and of its various organs, and upon the causes of parenchymatous and fatty degenerations. The arguments advanced in support of the view that all of the characteristic symptoms of fever are directly dependent upon the increase of temperature and that high temperature is the chief source of danger, are derived no less from experimental pathology than from clinical observations.

Three methods have been employed to determine the effects of increased temperature in fever: one is to study the effects of external heat upon man and animals; the second is to examine in different fevers and in different cases of the same fever the relation of the temperature to the other symptoms, and to the general condition of the patient; and the third is to note the influence of reduction of temperature upon these symptoms. Each method has its limitations.

The condition produced by exposure to external heat, even if it be called thermic fever, is something quite different from ordinary fevers, and we cannot transfer the results obtained by this method directly to the explanation of febrile phenomena. On the other hand, in the clinical study of fevers it is very difficult and often an arbitrary matter to separate the effects of increased temperature from those of other factors nearly always present. Hence we find no agreement of opinion among physicians as to what symptoms or lesions in fever are referable to the heightened temperature, and what are due to infectious or other conditions often present. Even the frequent or constant association of certain symptoms with elevated temperatures and their subsidence or disappearance by reduction of temperature do not justify us in inferring that the high temperature is the cause of the symptoms, for both may be coördinate effects of the same cause, and the so-called antipyretic treatment may influence other conditions as well as the temperature. As has been frequently said of late, the high temperature may be rather an index of the severity of the disease than a source of danger in itself.

In hyperpyrexia and in many cases of insolation there can be no doubt that the high temperatures, as such, are the main elements of danger. But both of these conditions have important points of distinction from ordinary febrile processes. In hyperpyrexia there is probably almost complete paralysis of heat regulation, and we shall find that an analogous condition with similar dangers sometimes develops in animals artificially heated. The cases of insolation in which the high temperature is so dangerous are analogous to the condition which may be produced in animals by brusque elevation of the bodily temperature through exposure to heat, and which

[1] Liebermeister: Volkmann's Sammlung, No. 31, p. 240.

differs from that resulting from more gradual increase of temperature.

The most direct way of determining the influence of heat upon the body is to raise the internal temperature by the external application of heat. Here we are not disturbed by the presence of other factors, such as infection, which render doubtful so many of the conclusions derived from clinical observations as to the effects of high temperatures. For the solution of many problems it is evidently irrelevant whether the source of heat be within or without the body.

These experiments, if properly conducted, are calculated to shed much light upon many questions relating to the effects of febrile temperatures.

According to the testimony of all experimenters, a mammalian animal, artificially heated, dies when its internal temperature reaches 111.2° F. (44° C.) or 113° F. (45° C.). Death is preceded by convulsions and immediately or soon after death rigor mortis appears. At the moment of death the irritability of the heart and muscles ceases. Death seems to be due to heart paralysis, and the cause of this is usually set down as heat rigor, but this is not probable as death generally occurs at a temperature several degrees below that at which rigor of the heart muscle appears.

It is generally argued that temperatures several degrees below those which are fatal must exert toxic effects, and this conclusion seemed to be substantiated by the majority of experimenters, who found that animals whose temperatures were artificially raised to 105° F. (40.5° C.) or 107° F. (41.7° C.), or even to a lower point, manifested signs of illness. These latter results, however, are opposed to those obtained by Rosenthal, and especially Naunyn,[1] who points out that the conditions in most previous experiments were not favorable, as the animals were generally placed in small, dark, poorly ventilated metallic boxes. Naunyn succeeded in keeping a rabbit alive for thirteen days with an average temperature of 106.7° F. (41.5° C.).

Following Naunyn's example, I have had constructed a wooden box, three feet long, two feet high, and two feet broad, which fits closely within a double-walled galvanized iron box. The wooden box is provided with a perforated movable bottom, which allows the urine to escape. The space between the two walls of the iron box measures three inches across, and is filled with water, which, therefore, surrounds the inner box on all sides, except at the top, which is left open. The dimensions of this apparatus are somewhat larger than those of Naunyn's heating-box. A folded woollen blanket was drawn over each end of the box, so as to leave uncovered at least one-third, and generally more of the top. A rose burner placed underneath served to heat the box. At first a thermo-regulator was employed, but this was found unnecessary, as the temperature of the room varied but little, and there was no difficulty in keeping a sufficiently constant temperature in the box. The experiments were made upon rabbits, and only a single one was placed in the box at a time. Corrected thermometers were used. The temperature of the box was taken a short distance above the bottom, and that of the rabbit at a depth of three to four inches in the rectum. This occasion does not seem an appropriate one to describe these experiments in detail with their protocols. I shall take another opportunity for that, and at present give only a general account of those results which relate to the subject before us.

In the box described I have succeeded in keeping for three weeks two large black rabbits, the one with an average rectal temperature of 107.3° F, (41.8° C.), the other with an average temperature of 106.6° F. (41.4° C.). The rectal temperature fluctuated usually between 105.5° F. (40.8° C.) and 108° F. (42.2° C.), scarcely ever sinking below 105° F. (40.5° C.), but occasionally rising as high as 109.5° F. (43.1° C.). The temperature of the box varied between 96° F. (35.5° C.) and 106° F. (41.1° C.). The rabbits lay most of the time stretched out, breathing very rapidly. They took their food greedily, and did not appear ill. At the end of the experiment the first animal was removed from the box, and appearing perfectly well for ten days afterward it was used for another experiment; the second animal was killed at the end of the experiment, and presented marked fatty degeneration of the heart, liver, and kidneys. Both animals lost weight while in the box.

In these and similar experiments the rabbits were given only moist, green fodder, and were allowed to drink plenty of water, which they took eagerly. It is important for the success of the experiment that the temperature should be gradually, and not suddenly, raised.

Different rabbits offer varying degrees of resistance to the effects of high external temperature. It has seemed to me that black and gray rabbits surpass, in this respect, white rabbits. The same temperature of the box does not produce, in all cases, the same rectal temperature in different animals, or in the same animal at different times No factor is of more importance in determining the effects of external heat than the animal's power of temperature regulation. Failure of this power is manifested by a sudden rise of internal temperature, which may quickly attain a point incompatible with life. This event may take place without any alteration in the box temperature. The degree of internal temperature at which this paralysis of heat regulation occurs varies in different animals. One may be able to hold his temperature, for a short time, from further rise at as high a point as 109.5° F. (43.1° C.); in another, after the temperature has reached 107.6° F. (41.5° C.), there may occur, without any change in the box temperature, a sudden, and often fatal, elevation of temperature. In general, temperatures between 108° F. (42.2° C.) and 109° F. (42.8° C.) may be regarded as critical temperatures for these animals. One is forcibly reminded by these sudden and dangerous elevations of temperature of the occurrence of hyperpyrexia in certain fevers of human beings, and there is reason to believe that this, too, is caused by paralysis of heat regulation.

It seemed to me of some practical interest to determine what effect upon the rabbit's power of resisting high temperature is exerted by exhausting influences, particularly by anæmia. For this purpose rabbits which had been moderately bled, and others which had been used for some other experiments, were placed in the hot box. It was found that these animals are, although not without occasional exceptions, unmistakably less resistant to the effects of high temperatures than are robust animals. They succumb sooner, and at lower box temperatures.

We may now consider what inferences may be drawn

[1] Rosenthal: Zur Kenntniss der Wärmeregulirung u.s.w., Erlangen, 1872. Naunyn: Archiv f. exp. Path. u. Pharm., Bd. 18.

from these and similar experiments as to the effects of high bodily temperatures. It seems clear that a considerable part of the current arguments based upon experiments concerning the injurious effects of high temperatures must be revised in the light of Naunyn's experiments, and of those which I have briefly related. Because an animal may be killed by raising its temperature to 111° F. (43.9° C.), or 113° F. (45° C), it does not follow that an increase of temperature up to within 4° or 5° F. of this fatal point involves danger to life, or even any serious disturbance of the functions of the body. Although experiments in hot-air chambers show that in man brusque elevations of the temperature by only a few degrees give rise to serious symptoms, Krishaber[1] found that by habituation his temperature could be raised to 106.5° F. (41.4 C.), or 107.2° F. (41.8), without much discomfort. As small animals generally succumb more readily than large ones to artificial heating, it is not likely that the power of resistance in human beings is less than that found to exist in rabbits, and there is reason to believe that it is greater.

We cannot transfer directly to human beings the highest temperature at which we found rabbits can exist without serious discomfort save increased respiration. A rabbit's temperature is normally considerably higher than that of man, and apparently slight causes suffice to produce marked fluctuations. The normal rectal temperature of the rabbits used in my experiments was generally between 102° F. (38.9° C.) and 103° F. (39.4° C.). The highest average temperature at which a rabbit was kept for three weeks in the hot box exceeded, therefore, by 4° to 5° F. the average normal temperature. Such an increase would not correspond to a high febrile temperature in man. We can, however, with equal, and probably greater propriety, compare this temperature with that at which death surely occurs in a condition bordering on heat rigor. This temperature (111° to 113° F.) is probably about the same for man as for rabbits and other mammalians. From this point of view the inference may be drawn, although, of course, with much reserve, that human beings may tolerate temperatures of 107° F. (41.7° C.), or even higher, for a considerable time. This inference is supported by clinical observations, especially in cases of relapsing fever.

As already pointed out, the condition produced by artificial heating is not directly comparable with that in fever. In the former the loss of heat from the body is reduced to a minimum; the superficial temperature is three or more degrees higher than the internal, so that the average temperature of the whole body is higher than in fever with the same internal temperature. We cannot say, therefore, but that man may tolerate considerably higher internal temperatures when the elevation is brought about under conditions in which the discharge of heat is not checked than when the temperature is forced up by stopping heat dissipation. This would be proven if confidence could be placed in the enormous elevations of temperature reported by Teale and others. In these cases, however, even if authenticated, it is probable that heat is abnormally distributed in the body, and we cannot infer that the internal temperature is uniformly raised to such paradoxical heights.

It is of the utmost importance to bear in mind that, as my experiments have shown, not only do animals differ in their power of tolerating high temperatures of the body, but this resistance may be weakened by various depressing causes. Nothing would be more irrational than to conclude, because one individual in a certain condition can tolerate very high temperatures, another in a different condition possesses the same power. In fevers we have various factors, particularly infection, which we may well believe can lower the tolerance of high temperatures. That in some fevers, particularly relapsing fever, this does not appear, or only partially, does not disprove that in another fever, such as typhoid or pneumonia, the system may be placed by other factors of the disease in such a condition that even moderately high temperatures are injurious. It does not seem to me proper in these cases to lay, as some seem inclined to do, the sole stress upon the element of infection. This is in all likelihood the determining factor, but the practitioner cannot shut his eyes to the possibility that under its influence the high temperature, as such, is a source of danger to his patient.

With these restrictions I shall surely not be misunderstood when I assert that temperatures which are ranked as high febrile temperatures do not in themselves, independently of other factors, exert any such injurious influence as has been usually attributed to them.

Our attention up to this point has been directed to the effects of high temperatures upon the general condition of the body. It is of importance for the proper understanding of fever to determine the influence of heat upon the structure and functions of the various organs. To what extent can the febrile disorders of respiration, of circulation, of secretion, of nutrition, of innervation be attributed directly to the elevated temperature? These are questions which can be answered better by experimental methods than by clinical observation, for the latter has to deal with the effects of heat complicated by other circumstances whose influence cannot be accurately determined. The one method, however, should be made to control the other.

The most striking immediate effect of heat upon an animal is increased frequency of respiration. When a dog or a rabbit is placed in an atmosphere of 100° F. (37.8° C.), it at once begins to pant and the respiration may run up to 150° or more. The causation of this increased respiration, to which the name heat dyspnœa has been applied by Ackermann,[1] has been repeatedly investigated. Goldstein[2] in Fick's laboratory found that by applying to the carotid arteries tubes through which hot water is flowing the respirations may be greatly increased in frequency. Goldstein's experiment is usually cited as the crucial one, showing that the increased breathing is due to the effect of the heated blood upon the respiratory centres in the medulla oblongata. Sihler,[3] working in the Biological Laboratory of the Johns Hopkins University, however, has demonstrated that the increased respiration of an animal exposed to heat is due to two causes, warmed blood and stimulation of the skin by the heat, and that probaby skin stimulation is the more important factor. Some of Sihler's criticisms of Goldstein's experiments have been met by Gad and von Mertschin-

[1] Krishaber: Gaz. Méd. de Paris, 1877.

[1] Ackermann: Deutsches Archiv f. klin. Med., Bd. ii.

[2] Goldstein: Würzburger Verhandl., 1871.

[3] Sihler: Journal of Physiology, vol. ii., and Studies from the Biological Laboratory, Johns Hopkins University, Baltimore, vol. ii.

sky,[1] who have made it evident that increased temperature of the blood stimulates the respiratory centres, or increases their irritability. Section of the vagi does not check heat dyspnœa, so that this does not result primarily from the action of the increased temperature upon the terminal pulmonary expansion of these nerves. The removal of afferent impulses from the skin by section of the spinal cord does, however, exert so marked an influence that it cannot be doubted that heat stimulation of the skin is an important element in the causation. I have convinced myself of the correctness of Sihler's explanation by observing that in the hot box at temperatures of 90°–95° F. (32.2°–35° C.) rabbits often preserve their normal temperature, and still their breathing is markedly increased, and that, on the other hand, upon taking them out of the box the respirations may sink before the internal temperature begins to fall. In one striking experiment the respirations fell immediately to about normal upon removing from the hot box a rabbit whose skull had been trephined on each side of the median line, and the optic thalami punctured with a needle.

We cannot transfer directly to human beings the results of experiments on heat dyspnœa in animals, because in the latter respiration has a far more important function in temperature regulation than in the former. To keep cool a dog pants under circumstances when a man sweats. As heat regulation is largely influenced by the cutaneous temperature, it is not improbable that in man heat stimulation of the skin is less prominent than the warmed blood acting upon the respiratory centres in increasing the frequency of respiration in fever.

Inasmuch as disturbances of the heart and of the circulation in general are among the most important symptoms of fever it is natural that much attention should have been given to the study of the effects of heat upon the organs of circulation. The supposed injurious effects of prolonged high temperatures in fever have been usually attributed especially to some toxic action of heat upon the heart.

That the pulse-rate is quickened by artificially heating rabbits and dogs has long been known. The positive demonstration that this acceleration is due to the direct action of the heated blood upon the mammalian heart itself was first given by my colleague, Prof. Martin.[2] By conducting through the dog's heart, isolated physiologically by the ingenious method which he devised, Martin proved that the heart "beats quicker when supplied with warm blood and slower when cold blood is supplied to it; also, that the rate of beat depends much more upon the temperature of the blood in the coronary arteries than on its temperature in the right auricle or ventricle." These experiments make it unnecessary to recur to any action of the heated blood upon extrinsic cardiac nerves or nerve centres in order to explain the quickened pulse of fever. Moreover, Fick[3] found that the nervous centres of the heart and bloodvessels are unaffected by heating the blood flowing through the carotid arteries in the manner adopted by Goldstein in his experiments on heat dyspnœa.

[1] Gad and v. Mertschinsky: Virchow u. Hirsch's Jahresbericht, 1881, Bd. i. S. 197.

[2] Martin: The Direct Influence of Gradual Variations of Temperature upon the Rate of Beat of the Dog's Heart, Philosophical Transactions of Royal Society, part ii. 1883.

[3] Fick: Pflüger's Archiv, Bd. v.

Prof. Martin has kindly permitted me in this connection to mention certain unpublished results of experiments which he is now conducting upon the effects of heat and cold on the isolated heart. As these results are pertinent to our subject I gladly avail myself of this privilege. The table of an experiment which I have examined shows that the isolated cat's heart beats regularly and more and more rapidly as the temperature of the blood is gradually raised to 111.2° F. (44° C.). At this point the beats become irregular, but are restored to their normal rhythm by feeding the heart with cooler blood. The temperature of about 111° F. (43.9° C.) appears to be a critical one for the isolated heart. Above 111.2° (44° C.) to 113° (45° C.) the pulsations become slower instead of quicker as the temperature is raised. At 122° F. (50° C.) the heart's action ceased, but the heart was made to beat again by supplying it with cooler blood, showing that the cessation was not due to heat rigor. This interesting experiment teaches among other things that very high temperatures may produce results differing not only in degree but also in kind from those of temperatures only a degree or two lower.

We may consider it then established that increased frequency of the pulse in fever is referable to the direct action of the warmer blood on the nervo-muscular substance of the heart itself. Clinical observation of cases of fever makes it evident that there may be and often are present other circumstances which influence the rapidity of the heart's pulsations, circumstances which in themselves may slow or may quicken the pulse. Large series of statistics, therefore, are required to bring out the ratio between the pulse-rate and the temperature in fever and even then for any given temperature the maximum and the minimum pulse-rates lie so far apart that the statement of the average increase in the frequency of the pulse for each degree of rise of temperature, such as has been computed by Liebermeister, has very little value.

Not only is the pulse-rate quickened in fever, but there are often other and more serious circulatory disturbances. In the fever produced in rabbits by injection of the swine plague bacillus I find a reduction of the blood-pressure measured in the carotid artery, and others have obtained similar results in the artificial fevers of animals. The determination of the blood-pressure in fevers of human beings by means of Basch's sphygmomanometer has given, in the hands of different experimenters[1] such contradictory results, that no conclusion can be drawn, unless it be the very probable one that the blood-pressure varies, being sometimes high, sometimes low, and sometimes normal in fever. That the arterial tension is often reduced is made evident by the marked dicrotism of the pulse wave. This dicrotism, however, characterizes particularly septic and typhoid types of fever and is absent during the chill of intermittent fever and often in exanthematous and some other fevers, so that we cannot consider the blood-pressure and arterial tension as having any such definite relation to fever as does the pulse-rate.

It has been observed by Paschutin, Senator, and Mendelson,[2] that the blood pressure rises with increasing

[1] Von Basch, Zadek, Arnheim, Wetzel.

[2] Paschutin, Ludwig's Arbeiten, 1873. Senator, DuBois-Reymond's Archiv, 1883, Supplement-Band. Mendelson, On the Renal Circulation during Fever, Amer. Journ. Medical Sciences, October, 1883.

bodily temperature, produced by exposure to heat. Mendelson found that the pressure begins to sink as the temperature approaches a point incompatible with life. In these experiments the animals were subjected to rapid elevations of temperature. I find that when a rabbit is gradually and cautiously heated in the hot box used in my experiments, the rise of temperature is less marked, and may not surpass even for temperatures of 107° F. (41.7° C.), the normal limits of variation which may be found in the blood-pressure of the same animal examined at different times. These measurements, however, are not very conclusive, for, as has been already remarked, rabbits which have been operated upon do not stand well artificial heating.

I have observed that the rhythmical contractions of the bloodvessels of the rabbit's ear are feeble or absent when the temperature is much elevated by artificial heating; whereas, it will be remembered that in experimental septic fever these contractions are irregular and exaggerated.

Upon the whole, I think that we are justified in concluding that the variations of arterial tension in fevers are much less dependent upon increased temperature than upon other factors, such as infection.

But the corner-stone of the doctrine which teaches that a chief source of danger in fevers is the elevation of temperature, is not the effect of increased temperature upon the pulse-rate or the arterial pressure, but it is the belief that prolonged high temperature exerts a directly paralyzing influence upon the heart. The main support of this belief is not the admitted fact that extremely high temperatures paralyze the heart, for these critical temperatures lie far above the ordinary high temperatures of fever, and in a region where all admit the dangers of the excessive internal heat. The stately superstructure has been built up chiefly on the basis of experiments showing that when the internal temperature of animals has been maintained for some time at a high point by exposure to external heat, parenchymatous or fatty degeneration of the heart muscle ensues. It is true that all experiments are not in accord upon this point, and that, as a rule, pathological anatomists have not given adherence to the doctrine that parenchymatous degenerations are chiefly dependent upon high temperature, still this doctrine has gained a wide acceptance among clinical men, and is advocated with especial force by Liebermeister. I have, therefore, thought it desirable in my experiments to give especial attention to this question.

Among previous experimenters on artificial heating of animals, Iwaschkewitsch, Wickham Legg, and Litten may be mentioned as finding parenchymatous or fatty degeneration of the heart, liver, and kidneys; and Walther, Obernier, and Naunyn as obtaining only negative results.

It is not necessary here to enter into a criticism of these different experiments, which are of very unequal value. Litten's[1] experiments on the one side, and Naunyn's[2] on the other, appear to be the most carefully conducted. Litten, whose experiments are those usually cited, kept guinea-pigs in a double-walled metallic box which was at a constant temperature of 98.8° F. (37° C.) with dry air, or of 96.6° F. (36° C.) with moist air. He never failed to find fatty degeneration at the end of thirty-six to forty-eight hours. The liver was first affected, and then the heart and kidneys, which become fatty by the second or third day. The animals did not survive longer than five or six days, and by that time the fatty degenerations had reached an extreme degree. Von Recklinghausen[1] urges with apparent justice against these experiments that the enforced inactivity of the muscles and the imperfect ventilation may have had as much to do in causing the degenerations as had the high temperature. These objections find support in the experiments of Naunyn, who, making use of a much larger and better ventilated heating box than Litten, failed to find any parenchymatous or fatty degeneration in his rabbits after they had been exposed for two weeks to higher temperatures than Litten employed.

As my experiments confirmed in other respects Naunyn's results, I was quite unprepared to find that my rabbits, after a variable period of artificial heating quite constantly presented fatty degeneration of the heart, liver, and kidneys, and sometimes of the diaphragmatic and intercostal muscles. I never found the degeneration at so early a period as did Litten. It was not until the rabbit had been kept for at least a week with an average rectal temperature of 106° F. (41.1° C.) that the degeneration was noticed, and then only in moderate degree. The higher and the less fluctuating the internal temperature, the more certain was the degeneration to appear. I could reckon upon obtaining rabbits with well-marked fatty degeneration of the heart, by keeping them for ten days with a rectal temperature between 107° F. (41.7° C.) and 108° F. (42.2° C.). I am not prepared to account for the discrepancy in this respect between Naunyn's and my experiments. The box used was larger than that employed by him, and every care was taken to keep it well ventilated by leaving at least one-third and often one-half of the top open.

I do not think that my experiments altogether do away with the force of von Recklinghausen's criticisms. The fact that in these experiments the degeneration made its appearance at the end of a week or ten days, and in Litten's in forty-eight hours, to say nothing of Wickham Legg finding it at the end of twelve hours, would suggest that if the rabbits were heated in a still larger and better ventilated apartment, the degeneration might not occur at all, or might be deferred to a much later period. In my opinion, however, even if full allowance be made for this line of argument, we must still admit that prolonged high temperature is a factor in the causation of fatty degeneration.

That it is not the sole factor no one can doubt. As is well known, fatty degeneration is produced by anæmia and by a variety of poisons, and even in fevers most pathologists are convinced that it bears a closer relation to the kind and the degree of infection than it does to the height of the temperature. It is more frequently absent than present in pneumonia, even where there have been symptoms of heart failure.

The kind of degeneration present in my rabbits was fatty and not parenchymatous. Probably all who make many post-mortem examinations will agree with von Recklinghausen, that altogether too liberal use has been made of the diagnosis of parenchymatous degeneration,

[1] Litten, Virchow's Archiv, Bd. 70. [2] Naunyn, op. cit.

[1] Von Recklinghausen: Handb. d. Allg. Pathologie d. Kreislause u d. Ernährung, p. 512, Stuttgart, 1883.

and not sufficient account has been taken of the anatomical changes of the parenchyma produced by post-mortem chemical changes, such as acid formation, etc.

In order to determine what influence is exerted by infection combined with high temperature, I inoculated a rabbit, which had been in the hot box for four days, with the bacilli of swine plague. These bacilli, if not identical with, are closely allied to those of rabbit septicæmia, and are extremely virulent for rabbits. In thirty-six hours the animal, which had remained at a high temperature in the box, was dead, with characteristic lesions of the disease, and the most extreme fatty degeneration of the heart and other organs was found. As in other experiments the degeneration had not made its appearance at this early date, there can be no doubt that the infection was an important element in the causation. That it received powerful support in the high temperature, however, is proven by the fact that little or no degeneration of the heart is observed after infection with this organism when the animal is kept at ordinary temperatures.

Admitting, then, that high temperature aids in the causation of fatty degeneration of the heart in fever, the question arises, What do we know of the effects of this degeneration upon the functions of the heart? I will say nothing of the growing tendency to transfer a large part of the classical symptomatology of Quain's fatty heart to other conditions, particularly to disease of the coronary arteries and chronic myocarditis; we are concerned at present only with the occurrence of this degeneration in fever. Have we not been somewhat hasty in assigning to degenerations of the heart's muscle so large a share in the production of heart failure in fevers? One cannot look at a muscular fibre in which the striated substance is all replaced by fatty globules, and suppose that its functional activity was unimpaired; but into what serious errors should we fall if we attempted to deduce from the anatomical changes in the liver cells or the renal epithelium the corresponding functional disturbances? Certain it is that symptoms which are usually considered those of heart failure are often enough present in fevers without finding at the autopsy any degeneration of the heart; and, on the other hand, such degeneration may be discovered without any history of these symptoms, although, of course, the two are often associated.

In the face of these doubts it seemed desirable to determine, if possible, experimentally the damage inflicted upon the cardiac functions by the presence of fatty degeneration of the heart muscle. That this degeneration may exist without apparent injury is rendered probable by the fact that a rabbit which has been kept for three weeks in the hot box at a high temperature, and in which there is every reason to suppose that fatty degeneration has occurred, may present no symptoms of heart paralysis, and when removed from the box appear and remain perfectly normal. An instance has already been mentioned where one rabbit at the end of three weeks was killed and presented marked fatty degeneration of the heart; and another, which had possessed a higher average rectal temperature, was removed from the box at the end of the same period, and appeared for ten days perfectly normal, when it was used for another purpose. That this degeneration can be recovered from is, moreover, rendered probable by clinical experience, and is universally admitted.

Once in teasing out, in a warm room, a bit of fatty heart muscle from one of the rabbits, I made a curious observation. Near the edge of the cover glass, where there was a slight current in the physiological salt solution, rhythmical contraction was observed in a group of muscle-fibres. This interesting spectacle could be watched under the microscope for ten minutes. These contracting fibres were filled with fatty globules, and only here and there, and then indistinctly, could any trace of striation be detected. This observation teaches that a fatty degenerated muscular fibre is capable of contraction, but, of course, warrants no further conclusions.

Far more important than any inferences which can be drawn from such observations is the measurement of the actual blood-pressure in animals whose hearts have undergone fatty degeneration. This I have done in three instances. The rabbit was removed from the box at the end of ten days to two weeks, and the pressure in the carotid artery was measured by a mercury manometer attached to Ludwig's kymograph. In no instance was the blood-pressure found to be lower than that normally present in rabbits. In an experiment recently performed, the average pressure was 125 mm. of mercury; the pulsations were regular; the heart responded to stimulation of the vagi in an entirely normal manner. After such stimulation the pressure in one case rose to 176 mm. of mercury. Immediately after the measurement of the blood-pressure the rabbit, as in the previous instances, was killed, and marked fatty degeneration of the muscular fibres was found. In many of the fibres the striation could not be made out, and only fatty globules were visible; in others, which also contained fatty granules, the striation was distinct. The degeneration involved the whole muscular substance of the heart. A similar appearance in a human heart would be considered to indicate well-marked fatty degeneration.

These experiments show that a rabbit's heart which has undergone marked fatty degeneration from exposure to heat, may perform its functions, to all appearances, and for the time being, in a perfectly normal manner.

There is at least one consideration which should make us cautious in drawing far-reaching conclusions from these experiments. There are diseases of the heart—I need only refer to lesions of the coronary arteries—in which the functions of the organ are performed for a longer or shorter time, apparently in a perfectly normal way, and then heart failure suddenly appears. It is probable that here too the blood pressure would be found normal at a certain period of the disease, and still it would be an error to suppose that the lesion does not damage the heart.

Whatever force there may be in this analogy, I still think that these experiments, as well as careful pathological and clinical observations, necessitate some revision of the current opinions concerning the significance of fatty degeneration of the heart in fever.

So much time has been devoted to a consideration of the effects of heat upon the respiration and the heart that the limits of the present lecture will permit hardly more than a summary of the effects exerted by heat upon other functions and organs of the body. On account of the great clinical importance of the subject it seemed desirable to treat with especial fulness the influence of increased temperature on the heart.

What part has increased temperature in producing febrile consumption of tissue? In the first lecture men-

tion was made of Pflüger's experiments showing that animals with elevated temperature, produced by exposure to heat, absorb more oxygen and excrete more carbonic acid than at the normal temperature. This is in conformity with the general law that within certain limits cell activity is more energetic at high than at low temperatures. It was also shown in the first lecture that only a comparatively small part of the increased oxygen absorption and carbonic acid elimination in fever can be referred to the immediate effects of high temperature. It has not been demonstrated that the respiratory gases in human beings are increased by artificial elevation of temperature. Indeed, Voit[1] was unable to find any such effect of increased temperature in human beings on the respiratory gases as both he and Pflüger observed in animals.

Inasmuch as increased disintegration of nitrogenous material is such a prominent disorder in fever, much attention has naturally been given to determining how far this can be explained by elevated temperature. That it cannot all be so explained is proven by the interesting observation of Sydney Ringer, that excessive elimination of urea antedates the rise of temperature in intermittent fever, and Naunyn has found the same to be true of the septic fever of dogs. The experiments which have been made to determine the influence of artificial heating on the amount of urea excreted by man and by animals have yielded contradictory results. A number of these experiments are of little or no value, because no attention was given to establishing beforehand nitrogen equilibrium. Schleich's[2] experiments may be mentioned as, perhaps, the most accurate of those showing an increase in the urea excreted under the influence of exposure to heat. The more recent experiments of Simanowsky[3] were made in Voit's laboratory upon a dog with especial precautions as to the establishment of nitrogen equilibrium. He failed to find any increase in the excretion of urea as the result of exposure to external heat. While then this question must be left at present *sub judice*, there can be no doubt that only a part at least of the excessive disintegration of nitrogenous substance in fever can be assigned to the influence of the increased temperature.

The loss of weight exhibited by animals kept for a long time in a hot atmosphere is usually explained by the excessive evaporation of water from the body. In many of my experiments the rabbits were freely supplied with food and water, and still the loss of weight was very noticeable. I am inclined, therefore, to attribute to the increased temperature under these conditions a decided influence upon the consumption of tissue.

Senator[4] found the urine of rabbits artificially heated to contain more albumen than is ever found in the urine of healthy rabbits. This observation I have not been able to confirm on the rabbits in my experiments, and probably this difference is to be explained by the more rapid and intense heating employed in Senator's experiments. Senator explains the heat albuminuria by the rise of arterial pressure in the renal vessels, but this is not in conformity with the interesting experiments of Mendelson,[1] who found by means of Roy's oncometer that both in thermic and in septic fevers of dogs the kidney is anæmic, while the general blood-pressure is elevated.

No satisfactory explanation has yet been offered of the diminution of perspiration which distinguishes fever so strikingly from the condition produced by exposure to high external temperatures. Luchsinger's assertion that this is the result of lessened irritability of the sweat centres in the spinal cord remains to be proven, and at the best is not a satisfying explanation.

Bokai,[2] in a recent experimental research on intestinal peristalsis in thermic and in septic fevers, comes to the conclusion that the constipation of fever is to be explained by the heated blood stimulating the nerves inhibiting intestinal peristalsis. If this should be confirmed, then it would be proven that at least three symptoms of fever, the quickened respiration and pulse and the constipation, are direct effects of elevated temperature.

When we consider the important nutritive changes in the muscles accompanying increased thermogenesis, we shall be inclined to attribute in part, at least, to these alterations and the associated abnormal innervation, rather than to increased temperature, the muscular pains and weakness which form such an early and frequent complaint in many fevers.

The investigations hitherto published of changes in the blood produced by increased temperature within febrile limits are not of sufficiently definite and satisfactory nature to warrant any consideration on the present occasion.

Nor am I acquainted with any experimental evidence (save Bokai's work already mentioned) that increased temperature is concerned in the production of the digestive disorders of fever. It has already been said that the rabbits with high internal temperatures in the hot box ate greedily, but these voracious animals cannot be considered favorable subjects to test this question.

Especial emphasis has been laid by Liebermeister and those who accept his teachings upon the dependence of the nervous symptoms of fever, particularly the so-called typhoid symptoms, on the elevation of temperature. There is, however, abundant clinical evidence to disprove this doctrine. Reference need be made only to relapsing fever, and especially to the aseptic fever described by Genzmer and Volkmann, in which there is entire absence of the whole group of so-called nervous symptoms. Moreover, Liebermeister's opinion in this respect is not supported by adequate experimental evidence. Unless the temperature of the brain or of the entire animal be brusquely raised to a high point by coarse methods, no disturbance of the cerebral functions is noticed until the temperature reaches a critical point, beyond which further increase is likely to prove rapidly fatal. At this critical point the animal, which before has lain quietly, becomes very uneasy, and if the temperature rise higher it has convulsions and coma and dies.

I have endeavored to bring before you in this lecture the experimental evidence relating to the effects of increased temperature upon the general condition of the body and upon the functions of its various organs. I have given account, as briefly as possible, of some ex-

[1] Voit: Zeitschrift für Biologie, Bd. xiv.
[2] Schleich: Arch. f. exp. Path. u. Pharm., Bd. iv.
[3] Simanowsky: Zeitschr. f. Biologie, Bd. 21.
[4] Senator: Du Bois-Reymond's Archiv, 1883, Supplement-Band.

[1] Mendelson: Op. cit.
[2] Bokai: Archiv f. exp. Path. u. Pharm., Bd. 23.

periments which perhaps shed additional light upon this important subject. In the course of this lecture emphasis has repeatedly been laid upon the necessity of controlling the results of the experimental method by clinical observation, and here and there I have endeavored to institute this control. Further than this I shall not attempt to set before you the clinical evidence regarding the effects of increased temperature in fever. There are those here more competent than I to deal with this side of the question. We may feel assured that when all the facts are before us and are properly interpreted, there can be no lack of harmony between the results of experimental and those of clinical investigations.

ORIGINAL ARTICLES.

A CASE OF CONSERVATIVE CÆSAREAN SECTION, TERMINATING IN RECOVERY.

BY W. W. JAGGARD, M.D.,

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I DESIRE to place on record the following case of conservative Cæsarean section performed under the relative indication, and terminating in recovery, and, in passing, beg to call attention to certain points of practical interest in connection with the indication for, and the technique of the operation.

Dr. P. Dougherty, of Chicago, a short time since invited me to see in consultation a case of alleged contracted pelvis. We examined the patient in the third week of February last, and elicited the following history:

CASE.—Mrs. E. S., thirty-six years old, born in Hillesheim in the region of the Eyfel Gebirge, Rhenish Prussia, married in the United States, October, 1880, shortly after immigration. She had been a sickly child, unable to walk until her seventh year on account of *Doppelglieder—i. e.*, rachitis. During infancy she suffered from tuberculosis of the cervical glands, two depressed cicatrices being visible on the left side of the neck at the time of examination. Since her seventh year she has enjoyed robust health.

Her mother gave birth to four children, of whom three were females. All of these labors were normal. Of the patient's two sisters, one has had normal confinements, while the other has been invariably delivered with the aid of instruments.

First pregnancy; patient's first child delivered May 13, 1882. Shoulder presentation, right-scapulo-anterior position. Difficult delivery after version and decapitation. Puerperium normal.

Second pregnancy; delivery June 20, 1883. Same presentation and position as in first pregnancy. Prolapsus of funis. Version, extraction, forceps to the after-coming head. Septicæmia, puerperium six weeks.

Third pregnancy; delivery October 4, 1884. Same presentation and position as in two former pregnancies. Version, extraction, craniotomy on after-coming head. Puerperium normal.

Fourth pregnancy; induction of premature labor at the end of the seventh lunar month. Same presentation and position as before. Version and difficult extraction. Child survived the operation a few hours. Puerperium normal.

Fifth pregnancy; beginning of last menstruation June 1, 1887. *Status præsens:* The patient, of strong frame and well-developed muscles, is four feet seven inches high, and weighs one hundred and thirty-five pounds. Pregnant, near term; distance from ensiform cartilage to pubes forty-five centimetres [17½ inches]; from ensiform cartilage to umbilicus, twenty-two centimetres [8¾ inches]; circumference around umbilicus, eighty-seven centimetres [34 inches]. Shoulder presentation, right-scapulo-anterior position. Pelvic measurements:

Distance between anterior superior spinous processes	27 cm. [10½ in.].
Distance between iliac crests	27 cm. [10½ in.].
External conjugate diameter (Baudelocque)	14 cm. [5½ in.].
Distance from sacro-coccygeal joint to subpubic ligament (A. G. E. Breisky)	9 cm. [3½ in.].
Distance between the great trochanters	30 cm. [11.7 in.].
Pelvic circumference (Kiwisch)	85 cm. [33¼ in.].
Diagonal conjugate diameter	7.5 cm. [2.9 in.].
True conjugate diameter (estimated)	5.5 cm. [2.14 in.].

Diagnosis: Simple, flat rachitic pelvis, with so-called absolute contraction of the true conjugate diameter. Apart from the pelvis, the osseous system showed no marked signs of rachitis. There was no abnormal spinal curvature, antero-posterior or lateral, and the long bones were perfectly straight.

Indication.—Notwithstanding the fact that the pelvis was a typical example of the so-called absolutely contracted, simple, flat, rachitic class, the history of former deliveries demonstrated plainly that the obstacle to the escape of the child through the natural passages was only relative, and not at all insurmountable. To explain the woman's survival of former labors it is not necessary to invoke extraordinary skill upon the part of the medical attendants—in all twelve in number—nor unusual physical endurance upon the part of the patient, although both conditions were doubtless supplied. Both parents were undersized, with relatively small heads, and the children were of a size less than is common. Moreover, the after-coming head invariably presented, and the accommodation of the passenger to the passages was thus greatly facilitated. The case was clearly one in which the woman could be delivered with safety in all probability by version, extraction, and craniotomy.

On the other hand, the child was living and Cæsarean section offered the possibility of saving both mother and child, although, of course, with enormously increased maternal risk.

The question of the induction of premature labor, so late in pregnancy, was not considered for obvious reasons. In a word, the relative indication for Cæsarean section was presented.

The most important condition upon which this indication rests at the present time, is the consent of the woman, obtained without direct or indirect

coercion. Accordingly a plain, unvarnished statement of all the facts in the case was made to the woman. She was clearly and distinctly informed that by destruction of the child her life would be almost certainly saved, and that the attempt to save both lives by Cæsarean section would be attended by great danger to herself. After a week's deliberation she elected the Cæsarean operation. In reaching this conclusion, she was assisted by the Roman Catholic priest of the parish. This gentleman remarked that the pregnant woman was the aggressor, that she had made the contract of maternity; the child was passive and had made no contract. In strict equity, entirely apart from ecclesiastical considerations, the child's claims to life should be considered at least equally with those of the mother.

Of course, as an operator, I was more pleased to perform Cæsarean section than craniotomy. However, I cannot forbear to reiterate here certain convictions which must always come up for discussion in similar cases. These propositions, I beg to submit if the expression be not too harsh, not so much as matters of opinion, as matters of fact.

1. The necessary maternal mortality of craniotomy, performed under the conditions demanded in Cæsarean section, with the best instruments and adequate skill, in cases of the contracted pelvis with a *conjugata vera* of six to eight centimetres, is zero. The maternal mortality of the conservative Cæsarean section is still considerable. The shock incident to the operation, entirely apart from sepsis and the loss of blood, is an element of danger that can never be completely eliminated. It is, perhaps, needless to remark that the necessary degree of operative skill, the necessary experience in Cæsarean section, and other conditions are much more difficult to supply.

2. The consent of the patient, obtained without direct or indirect coercion, is an essential condition to the relative indication. The time will probably come when the woman shall not be permitted to elect as freely as she must be allowed to do at present.

3. The life of the adult woman, who has already contracted relations with society, is of incomparably greater value, as judged by human standards, than the problematical existence of an unborn babe. Moreover, the expectancy of life in such children is decidedly less than in children of normal birth. If the operation is performed, as in the case under consideration, before the objective changes of labor are noticeable, there is the risk of the premature interruption of pregnancy, of obviously serious prognostic moment with reference to the child. The necessary, early ligature of the cord deprives the infant of an average amount of blood of ninety-two grammes (Budin, Ribemont). The mother is seldom able, even if she were to be permitted, to suckle her child. Finally, the offspring of women affected with rachitis or osteomalacia, are frequently feeble, sickly, unable to resist the unfavorable influences of the environment, entirely apart from the effect of hereditary disease.

Operation.—The patient at once entered Mercy Hospital. The urine was examined and found to be normal. The only preparatory treatment consisted in a daily bath, in tepid water, with the liberal use of soap, which the woman's mode of life before admission rendered necessary.

In the selection of the time for operation I had determined, acting upon Schrœder's advice, to choose the latest possible moment before labor actually began. The advantage of an aseptic genital canal, daylight, adequate assistance, and the like far outweigh the danger of insufficient uterine contraction and retraction after evacuation of the viscus. Of course, one runs the risk—of moment to the child—of interrupting pregnancy some time before term. From the usual data—date of last menstruation, size and position of the uterus, abdominal measurements, length of the child measured by callipers (Ahlfeld), estimate of the size and weight of the child by palpation (Carl Braun)—it was only possible in this case to make a probable diagnosis of the time of gestation. I concluded the woman was in the last fortnight of pregnancy.

Early Tuesday morning, March 6th, she informed me that she would certainly fall in labor within the next twenty-four hours. She based her prediction upon dull pain referable to the lumbar and sacral regions, and beginning painful uterine contractions. She had been enabled to foretell her other confinements by similar sensation, and I was inclined to attach considerable importance in this case to subjective signs. The only objective change, indicative of impending labor, was a slight increase in the force of the intermittent uterine contractions; accordingly, the hour for operation was fixed upon at one in the afternoon. All precautions were taken with respect to the most thorough cleanliness and disinfection of the operator, assistants, patient, instruments, and environment. Dr. W. E. Casselberry administered the anæsthetic, ether. Dr. E. C. Dudley, Dr. Bayard Holmes, Dr. G. W. Whitfield, Dr. B. L. Riese, Dr. M. Scheuer assisted me in the operation. Dr. P. Dougherty and Dr. Charles Caldwell assumed charge of the babe. I take this opportunity to make my grateful acknowledgements to these gentlemen for their efficient services.

The woman was in excellent condition, cheerful, pulse and temperature normal.

The steps in the operation were: after evacuation of the bladder, incision through the linea alba, from the navel to a short distance above the pubes, as low down as was safe on account of the bladder. The diastasis of the recti muscles was well marked, and the peritoneum was incised without dividing much muscular tissue. No omentum nor intestines pre-

sented between the uterus and anterior abdominal wall.

The median line of the uterus coincided with the incision, and the usual manipulation to correct lateral version and axial rotation was unnecessary. Before making the incision, Dr. Holmes placed one hand on either side of the cut, and rendered the abdominal parietes tense enough to prevent the access of fluid to the peritoneal cavity. I incised the anterior uterine wall in the median line at a point a short distance above the os internum with a scalpel, and rapidly enlarged the cut in the direction of the fundus to the extent of thirteen centimetres [5 inches], with a blunt-pointed bistoury. The thickness of the uterine wall was about one centimetre [⅓ of an inch]. The placenta was implanted over the line of incision, and the first gush of blood was frightful. The placenta was quickly separated by the hand, the amnion ruptured, the child caught by the feet, turned and delivered without any laceration of the uterine incision. The amnion was not previously ruptured *per vaginam*, because I had confidence in the ability of Dr. Holmes's hands to keep blood and liquor amnii out of the peritoneal cavity. The child uttered a lusty cry upon its liberation from the cavum uteri. I had requested an assistant to insert the index finger into the upper and lower angles of the uterine wound, and bring it up close to the abdominal incision as an additional precaution against the escape of fluid into the peritoneal cavity. In the hurry of the operation this request was forgotten. After, or rather during the evacuation of the uterine contents, Dr. Holmes, by his hands over the abdomen, pressed the uterus through the abdominal incision. When the organ was entirely without the peritoneal cavity it was enveloped in hot compresses of sterilized gauze, while Dr. Riese brought the edges of the abdominal incision together and effectually prevented any intestinal protrusion.

Hemorrhage was trifling after the contraction and retraction of the uterine musculature, following the escape of the fœtus and envelope, and was now fully controlled by Dr. Holmes, who firmly compressed the lower uterine segment with the thumbs and index fingers of both hands. I did not apply the elastic ligature around the lower uterine segment before or after the uterine incision on account of the danger of paralysis of the structures at and below the point of compression, a danger to which Sänger (*Transactions of Ninth International Medical Congress*), Doléris, and others have recently called attention. The amount of blood lost during the incision need not be much greater without than with the elastic ligature. It need not be much more than the amount of blood in the uterus at the time the incision is begun, provided subsequent procedures are executed quickly. After the uterus is empty, uterine contraction and retraction, aided by digital compression, render the further loss of blood insignificant.

Twenty-one deep sutures were inserted, including all the tissues down to the mucosa. For the introduction of these sutures, I used the long, slender laparotomy needle of Thomas Keith. This needle passes with astonishing ease through the thick uterine wall, making a very small puncture, which is completely filled up with the suture material—in this case silk. After passing a finger through the cervix from above downward, the uterine cavity was irrigated with a five per cent. solution of carbolic acid, a bacillus containing ninety grains of iodoform placed within, and the wound closed. Union of the peritoneum over the line of the incision was effected by a continuous silk suture. When the two rows of sutures had been drawn taut, the uterine wound was accurately closed and perfectly dry. The uterus, in a state of normal retraction, was returned to the cavity of the abdomen. The toilet of the peritoneum was brief, as no fluid had escaped into the abdominal cavity, and the intestines had not at any time protruded. The abdominal incision was closed with interrupted silk sutures.

The duration of the operation was about one and one-fourth hours. From the extraordinarily simple *technique*, it would seem that the operation had been needlessly prolonged. But the uterine sutures were inserted deliberately and with care; then, too, time was occupied in securing retraction by the application of hot compresses.

The total amount of blood lost was not great, scarcely more than the average loss in normal labors. The chief element of danger lay in the suddenness of the loss, but no indication arose for the employment of transfusion, the apparatus for which was in readiness.

The shock from the operation was profound, but brief. The patient fully reacted within three hours. Her convalescence was uninterrupted. The pulse at seven the same evening was rapid, 120 beats to the minute, tense, and small. It became gradually less frequent, less tense, until at the expiration of the first week, it was normal. The temperature remained nearly normal, showing slight variations in the second week. These variations were attributed to several severe burns, suffered as the result of the injudicious application of hot bottles immediately after the operation. On the third day the audible escape of flatus was noted, and about the same time the patient began to void urine spontaneously.

Tympanites was notable by its absence throughout the recovery. On the fifth day, the bowels were painlessly evacuated after the exhibition of citrate of magnesia, for which the patient expressed a preference.

The patient did not vomit at all, not even when she was recovering from the anæsthetic.

The lochial discharge was slight, odorless, and ceased at the expiration of two weeks. Lactation was not established. In all former confinements, the milk secretion was abundant. The uterus underwent rapid involution, and at the time of writing the vaginal portion and vagina show few traces of recent pregnancy. The patient returned to the ward at the end of the third week.

The child was a small, but perfectly formed, and apparently mature male; weight, 3000 grammes [8¾ lbs.]; length, 49 centimetres [19$\frac{1}{16}$ inches]. The infant thrived on artificial feeding until the sixteenth day, when, after exposure to cold, it died suddenly, in a convulsion. The autopsy disclosed intense pulmonary congestion. Although the child

was apparently well nourished, it is not improbable that inanition was a predisposing factor. The death of the child was a matter of regret, apart from other considerations, on account of the possible unfavorable influence on the mother. She, however, calmly bore the loss, feeling happy that she had given birth to a living child capable of baptism.

The diameters of the fœtal head were:

Occipito-frontal .	11 centimetres	[4.29 inches].
Occipito-mental.	12 "	[4.68 "].
Biparietal .	8¾ "	[3.4 "].

The following extract from a letter recently received from Dr. Robert P. Harris, is of general interest in this connection:

"Your case makes 16 Sänger-Cæsarean sections for the United States, with 7 recoveries; and 165 for the whole Cæsarean list, with 63 women saved.

"I have 12 cases on record for the last fifteen months, with 6 women and 9 children saved; yours makes the thirteenth. There were 8 operations in 1887, all Sänger's but one, with 4 women and 5 children saved. I have 3 cases in already for this year: one each, for January, February, and March; 1 woman and 3 children saved."

2330 INDIANA AVENUE, CHICAGO, ILL.
March 31, 1888.

AN AID TO THE DIAGNOSIS OF HEMORRHAGIC PLEURISY.

BY FREDERICK P. HENRY, M.D.,
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IN a case of hemorrhagic pleurisy which I recently made the subject of a clinical lecture at the Philadelphia Hospital, I derived valuable diagnostic aid from Gower's hæmoglobinometer. Having excluded aneurism, tumor, and pericarditis with large effusion, I thrust into the pleural cavity a needle attached to a hypodermatic syringe, but much longer and stronger than the one employed for medicinal purposes. The fluid withdrawn appeared to be composed largely, if not solely, of blood, but on examining it carefully with the instrument referred to, I found that the amount of blood present in the exudation was really less than one per cent. In other words, if I removed fifty ounces of the fluid, the blood contained in it would not amount to half an ounce.

Of course, the blood withdrawn from the pleura was practically outside of the body before the aspiration, but the question to determine was whether, in case of rapid reaccumulation of the fluid, the amount of blood contained in it, supposing its proportion to remain the same, would be sufficient materially to lower the vitality of an already feeble individual. I was enabled to decide this question in the negative by means of Gower's instrument, and withdrew twenty-eight ounces of fluid with marked benefit to the patient. An enumeration of the blood corpuscles in the fluid was rendered impossible by the fact that these bodies were detected with great difficulty, being completely deprived of their coloring matter, and, therefore, identical in appearance with the phantom corpuscles (*Schatten*) described by Ponfick and others in cases of hæmoglobinæmia.

I am not aware that this test has ever before been applied in this manner, but there can be no doubt that it is a valuable aid to the diagnosis of hemorrhagic pleurisy.

ANGIOMA OF THE THYMUS GLAND.

BY T. P. BERENS, M.D.,
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J. C., aged sixty-three years, admitted to the surgical ward of the Philadelphia Hospital, November 29, 1887. His history was as follows: Mother and father died of senility, one brother killed accidentally, one brother and sister living and healthy.

The patient had always been a healthy man, moderate drinker of beer. Denied specific disease. For nineteen years had had leg ulcers, which he attributed to an attack of erysipelas. On admission he had two small, unhealthy ulcers, one upon each tibia. These, notwithstanding their treatment, grew worse, until the man developed marked constitutional depression, with many of the symptoms of pyæmia, dying ten days after admission.

The autopsy, performed under the supervision of Dr. Osler, revealed the following: The body is that of a good-sized, well-nourished man. Ulcers on each leg. Extensive hypertrophic nævus of lower lip; simple nævus of skin covering chin, right side of face, and right ear, extending also down the sternum, spreading and gradually fading into normal skin on either side of the chest. The tongue is much enlarged, and is noted to be in an angiomatous (cavernous) condition; immediately beneath it is a teat-like process, an inch in length and an inch at the base, where it seems to spring from the tongue above the frænum, and overlapping that structure. This also is a nævus. On removal of the sternum, in the anterior mediastinum the fatty tissues are permeated by a dense plexus of veins, which extends a short distance into the neck, forming a large nævoid tumor covering over the arch of the aorta, the roots of the great vessels of the neck, and the trachea up to the thyroid gland. On dissection this mass is seen to be composed of two distinct portions, and it represents a greatly enlarged thymus gland. The gland is enveloped in a capsule, under which some of the veins are large and prominent; on section the whole gland looks like a dense angioma; a plexus of veins closely surrounds the adventitia of the aorta. The thyroid is not enlarged; veins over it look full. Dr. Dock kindly cut sections which present those appearances commonly shown in cavernous angiomata, with but few if any traces of the normal glandular structure. In the heart the valves presented a condition of acute endocarditis, the rest of the organs being apparently normal.

A careful search was made through Virchow's *Archiv*, the Virchow-Hirsch *Jahresbericht*, *Transactions of the Pathological Society of London*, and many pathologies, without finding reference to such a condition of the thymus as is here described.

MEDICAL PROGRESS.

A Compound of Sulphuric and Carbolic Acids as a Disinfectant.—Laplace, who is studying the action of various antiseptics in Koch's laboratory at Berlin, reports in the *Deutsche medizinische Wochenschrift*, No. 7, 1888, his experiments with a mixture of crude sulphuric and carbolic acids, as follows:

His attention was drawn to the preparation of this compound by the insolubility of carbolic acid in water, and the need of overcoming this difficulty. To this end he combined crude carbolic acid with crude sulphuric acid; he found that equal parts of sulphuric with equal parts of carbolic acid (twenty-five per cent. in strength) produced a blackish, syrupy substance which remained soluble on cooling, and dissolved perfectly and rapidly in water. He further tested the potency of this compound as an antiseptic upon the bacilli of anthrax. His results showed that a four per cent. watery solution of this compound destroyed the virulence of anthrax in forty-eight hours.

Creolin, a recently tested antiseptic, is impotent in solutions of two per cent., to destroy anthrax; the acidified solutions of corrosive sublimate (1 to 1000) are potent against anthrax, but are not so available in practice. The cheapness of sulpho-carbolic acid, as Laplace styles this compound, and its availability for practical uses render these investigations of value.

Inflammation Masking Cancer.—Dr. Orecchia has recently called attention (*Gazzeta degli Ospitali*, March 4, 1888) to the frequent combination of inflammation with cancer in the same part, the graver disease being sometimes so obscured in this way as to be overlooked for a considerable time. He reports six cases in which this occurred. In one of these cancer of the larynx was supposed to be nothing more than perichondritis; in two, malignant disease of the jaw was mistaken for osteitis; in another periproctitis for some time masked a rectal cancer; parotitis concealed cancer or sarcoma of the parotid; and simple inflammation of the lip, an epithelioma. The possibility of malignant disease underlying what appears to be a simple inflammatory process should never be forgotten when the age and appearance of the patient, and the part affected, are such as to make the existence of carcinoma probable. Dr. Orecchia believes that in these cases the cancer is the primary disease, which, owing to the slightness of the symptoms, remains unnoticed till the supervention of inflammation calls attention to the part. In the stroma of all the tumors referred to, Dr. Orecchia found much extravasated blood, with abundance of leucocytes, of which there were also a great number in the neighboring tissues.—*British Medical Journal*, March 10, 1888.

Poisoning with Anilin Oil.—Dehio in the *Berliner klin. Wochenschrift*, No. 1, 1888, reports the case of a woman just recovered from childbirth, who took two and a half drachms of anilin oil with suicidal intent; the oil was used ordinarily to stain microscopic specimens. Coma, cyanosis, and profound depression followed. The skin was colored a grayish-blue, and intense hæmatogenic icterus followed, with extensive decomposition of the blood. Convalescence was tardy, recovery followed in about a month.

The Treatment of Convulsions in Children.—Simon has found the following, by rectal injection, most useful:

Chloral hydrat.	gr. 15.
Tinct. mosch.	gtt. 20.
Aquæ	ʒ 12½ to 15.

This may be given in two rectal injections, care being exercised to avoid violence, which may irritate or injure the bowel.—*Gazette Médicale*, March 3, 1888.

Multiple Salivary Calculi.—Dr. Nikolai J. Moiseeff, of Shtchizry, reports (*Proceedings of the Shtchizry Medical Society*, 1887, vol. i. p. 68) the case of a retired major-general, aged seventy, who sought his advice on account of incessant profuse salivation, pain on deglutition, and sublingual swelling of several years' standing. An oblong, hard elevation was found along the course of Bartholin's duct, which, even on slight pressure, gave the finger a distinct grating sensation. The duct was considerably distended along its whole course, and contained three salivary calculi lying in close contact. The nearest and smallest concretion, of the size of a big pea, was easily removed with forceps, but the other two could be extracted only after slitting up the duct, since they were considerably larger, especially the deepest one, which measured ¾ of an inch in length and ⅜ in breadth, and in addition was intimately adherent to the gland substance; it resembled a bird's bill in shape. When dry it weighed 33 grains, and consisted of a comparatively hard and compact nucleus, with a spongy, friable outer capsule. The smallest calculus was of a pyramidal form, of spongy consistence, and a grayish-yellow color; while the middle one was quadrangular, with rounded angles, and of a distinctly laminated structure. The total weight of the dried calculi was 38 grains. The wound healed well in a week. Dr. Moïsëëff has been unable to find another recorded instance of multiple calculi simultaneously present in the same salivary duct.—*British Medical Journal*, March 10, 1888.

The Reduction of Ferric Chloride by Pepsin and Allied Substances.—Duncan writes as follows on this subject to the *Chemist and Druggist*:

To the fact that the old tinct. ferri muriatis of the Edinburgh *Pharmacopœia* always contains ferrous chloride, is ascribed its alleged superior efficacy to the tincture of the present pharmacopœia. The following experiments which I have made seem to favor this view, and bear out a theory that ferric salts are reduced in the alimentary canal before absorption, and that their therapeutic value is in direct proportion to the amount of this reduction. Two grains of pepsin, B. P., were shaken up with 100 minims of water, and 10 minims of liq. ferri perchloridi, B. P. (free from ferrous salt), added, the whole being well shaken together. After standing for five minutes, 10 minims of two per cent. potassium ferridcyanide solution were added, when a blue color was

at once developed, showing that reduction of the ferric chloride had taken place. The same experiment was repeated with eight different samples of pepsin, all of which gave a similar result. With some, however, the reduction seemed greater than in other cases. It was also noticed that it increases with the length of time the pepsin and iron are left in contact. A control experiment was made in each case with pepsin and ferridcyanide, but in only one case was any blue color developed, and this most probably was due to an impure hydrochloric acid having been used in making the pepsin preparation. The ferridcyanide apparently does not undergo reduction, as the mixture does not become blue on the addition of a drop of pure ferric acetate solution. Possibly an albuminoid compound may be formed with the iron and pepsin, the iron being reduced during the combination; but I could find no evidence of such reduction with white of egg and ferric chloride. Similar results to these obtained with pepsin were given with ox-gall, pancreatin, papain, and saliva. The same experiments were made with liq. ferri acetatis and liq. ferri dialysatus. The former showed a slight reduction, the latter none at all, even after standing in contact with the pepsin for twenty-four hours. It is generally recognized that ferric acetate is less active therapeutically than ferric chloride, and dialyzed iron is least active of all, some therapeutists even denying that it has any efficacy, and this is apparently quite in accordance with the foregoing results. I do not regard these experiments as conclusive or complete, but the results are suggestive, and, as such, I record them, hoping that the investigation will be carried further.

The Treatment of Phlyctenular Keratitis in Children.—TROUSSEAU, in the *Revue Gén. de Clin. et de Thér.* of March 8, 1888, reports that he obtains good results from the following:

Vaselin	gr. 75.
Hydrarg. ox. flav.	gr. 3¾.

Once daily a bit of this ointment as large as a grain of wheat should be introduced into the eye. Three times daily compresses wet in a warm solution of boric acid should be placed upon the eyes for a quarter of an hour:

Acid. boric.	ʒ 3.
Aquæ	ʒ 11.

Trousseau classifies keratitis and its treatment as follows: Keratitis phlyctenular, superficial and vascular, and corneal opacities may be treated with the yellow oxide of mercury. Certain superficial keratites, corneal ulcers, and abscesses, call for eserine. In cases of parenchymatous keratitis atropia should be employed.

Ligature of the Superficial Femoral Artery, without Rupture of its Coats.—LUNN, at a recent meeting of the Clinical Society of London, reported the following interesting case:

C. W., æt. forty-five, widow, occupation a needle woman, was admitted May, 1887, into St. Marylebone Infirmary, Notting Hill, with pulsating tumor in the right popliteal space. No history could be obtained of a blow, strain, or syphilis, etc. The urine contained no albumen. She noticed a pulsating swelling about the size of an egg behind the right leg in the popliteal space. which gradually enlarged, and she was obliged to give up her work. The circumference of the pulsating tumor measured sixteen and a half inches. After giving a fair trial to different methods of treatment, and having failed to make any impression upon the aneurism, it remained only for me to advise the patient to submit to the usual operation of ligature of the superficial femoral artery, which she consented to do. The usual antiseptic precautions were taken; when the sheath of the artery was opened, the vessel wall was noticed to be a tallowy color, and not the usual pinkish tinge of health. Wishing to try Mr. John Smith's suggestion, and ligate the vessel with a clove-hitch, and as Mr. Ballance had had some experience of the clove-hitch in his as yet unpublished experiments on the carotids of horses and asses, I asked him to pass the kangaroo tendon, and occlude the vessel by means of a clove-hitch without rupturing the coats. This he did. The pulsation of the tumor was arrested immediately, and the woman made an uninterrupted and perfect recovery. The patient was kept in bed for seventy-two days after the operation. "The case is offered to this society as a contribution in aid of the advance of our knowledge of the surgery of our arteries."—*Medical Press*, February 29, 1888.

The Bacillus of Indigo.—The indigo of commerce is produced by the precipitation of a colorless glucoside contained in certain leguminous plants. This precipitation and the formation of the blue color of the indigo are effected, ALVAREZ believes, by a bacillus which causes a fermentation in the plant. This germ resembles that of pneumonia and of rhino-scleroma; and when injected into living organisms produces inflammation, especially of the genito-urinary tract.—*Revue de Clin. et de Thér.*, March 8, 1888.

A Physician's Experience in Self-treatment of Phthisis with Creasote.—The *British Medical Journal* of March 10, 1888, quotes the experience of a Russian physician as follows: The writer, who has been suffering from pulmonary and laryngeal tuberculosis for about two years, had tried the drug on himself in small doses (half a grain four or five times a day) some time ago, but without appreciable benefit. After perusal of the observations of Professors Sommerbrodt (*Berlin. klin. Wochenschrift*, No. 15, 1887) and Guttmann (*Deutsche med. Zeit.*, No. 42, 1887), however, he again began to take creasote in gradually increasing large doses, beginning with four grains a day, and reaching, in about two months, a daily dose of forty-four grains. There took place, fairly rapidly, an unmistakable and permanent improvement in his symptoms. Fever disappeared in a week; expectoration, cough, and dyspnœa steadily decreased to a considerable degree; laryngeal spasm, which had formerly occurred once or twice every month, ceased altogether. As regards the objective signs, however, there was only some diminution of dulness over a certain area, with complete disappearance of fine crepitant *râles*. As to tubercle bacilli in the sputum, they remained just as numerous as before the creasote treatment. The latter has lasted in all four months, during which period not less than four ounces and two drachms of pure creasote have been ingested. The drug must be taken in doses of about five grains four times daily, in the form of capsules (filled up *ex tempore*), after meals. With regard to

disagreeable after-effects Dr. Bogdanovitch observed in himself, when, by way of experiment, he took as much as twelve grains at a time, or twenty grains in the course of an hour, only giddiness, cardiac palpitation, small and accelerated pulse, general weakness, pallor, and anxiety; but all these toxic phenomena disappeared spontaneously and completely in about half an hour or an hour. On an empty stomach, however, he experienced epigastric uneasiness and pain even from small doses. Dr. Bogdanovitch resumed of late the use of creasote in order to study the effect of a six months' course.

Formulæ for Caustic Crayons.—De Sinéty gives the following formulæ, in *L'Union Médicale* of March 17, 1888:

Acid. carbolic. cryst.	gr. ¾.
Tannin.	gr. 60.
Glycerin.	gtt. 5.
Gum. tragacanth.	q. s.

These crayons should be about one inch long, and one-eighth of an inch in diameter.

The Contamination of Vaccine Matter by Micro-parasites.—Pourquier, in a recent communication to the Paris Academy of Sciences, reports his conclusions from several years' study of vaccine virus, which have led him to believe that ill results from such virus depend largely upon its contamination by a micro-parasite. He concludes that whenever the field of vaccination in a heifer presents an unduly inflamed and unhealthy appearance, the animal should no longer be used for that purpose. It may happen that but a portion of the surface used in an animal is affected; only the healthy pustules should then be chosen. Rigid antisepsis is imperative in instruments, the care of the animal and all concerned in its abode and treatment. If the micro-parasite enters a stable infecting a herd, the stable should be most carefully disinfected to limit and abolish the mischief.—*Gazette Hebdomadaire*, March 9, 1888.

Cocaine in Tooth Extraction.—The employment of certain salts of cocaine, and even the alkaloid itself, in dental surgery, has now received a fairly wide trial. It must be confessed that authorities here and abroad have arrived at very various results, some averring that cocaine is in all cases successful, while others have relinquished its use. To reconcile these discordant statements is at present quite impossible; we can only indicate the best means of applying this anæsthetic, and warn against its indiscriminate use. As a rule, from half a grain to a grain of the hydrochlorate dissolved in ten minims of water, and injected into the tissue of the gum by two punctures, one on the lingual and one on the buccal aspect of the tooth, insures a painless extraction. It is best to inject very slowly, and to wait from five to ten minutes after the completion of the last injection before extracting. However, in some persons cocaine fails entirely; nor is it possible to anticipate in whom it will succeed or in whom it will prove abortive. Simply painting the gum is valueless in tooth-extraction, except as a preliminary to the punctures of the hypodermatic syringe, for some persons are as much afraid of the pain incident to the pricking the hyperæmic gum as they are of the more severe operation. Although many persons experience no unpleasant effects from the injection of one grain, yet a certain proportion are painfully and alarmingly upset by its action. Syncope, extreme respiratory distress, feelings of complete prostration lasting for hours, vomiting, great nausea, facial paralysis, muscular paresis, and swelling of the tongue may ensue; thick utterance has been present in many cases; while vertigo, headache, and hallucinations are not uncommon sequelæ of cocaine given hypodermically. As a rule, these symptoms, although very alarming at the time, pass off in a few hours, but may persist for days. Cocaine sometimes loses its anæsthetic powers after the first injection. Many do not recommend cocaine for prolonged dental operations, preferring nitrous oxide gas, which is sure and safe, for short procedures, while others lean to a combination of the two agents.—*Lancet*, March 17, 1888.

A Good Domestic Disinfectant.—The *American Druggist* recommends the following solution:

Sulphate of zinc, commercial .	16 tr. oz.
Sulphate of iron, crystallized .	16 tr. oz.
Hypophosphorous acid (10 per ct.)	120 minims.
Oil of thyme	60 minims.
Naphthol	15 grains.
Water . . enough to make	5 pints.

Dissolve the two salts in four pints of boiling water, add the naphthol, and the oil of thyme, and shake or stir the mixture frequently while it cools. Then add the hypophosphorous acid, and make up the volume, with water, to five pints. Shake or stir the mixture frequently during a few days, then filter it through a wetted filter.

This solution will have a very handsome green color, and resist oxidation for a long time.

For use, it may be directed to be diluted with from five to ten parts of water, the strength depending upon the work it is expected to do.

Tannin Wool.—Benjamin Ward Richardson writes in the *Asclepiad*, No. 17, 1888, that in treating ozæna and other diseases attended with fetid odors, tannin wool turns out to be of great practical service. I had thought that the manufacture of this preparation had become a general fact; but as many medical friends have at various times written for the method, it may be of service to give the details here.

To make tannin wool, add to distilled water heated to 140° F. pure tannin up to saturation, stirring carefully all the time. When the water is saturated, add to it pure cotton wool, bit by bit, until all the solution is taken up by the wool. Lastly, put the saturated wool in an evaporating dish, and dry it slowly until it is quite dry. It is then ready for use, but must always be kept in a closed bottle. It must be neatly teased out before being used; but it is well to keep it in the rough state.

A good stock of tannin wool is at all times a useful thing to keep at hand. It is a ready styptic and possesses good antiseptic properties. It can easily be iodized by making an ethereal solution of iodine, saturating a portion of the cotton with the solution, and allowing the ether to evaporate. One grain of iodine to an ounce of the cotton is sufficient to make a very good specimen; and an iodized cotton made in this proportion is one of the best applications for bedsores that can be adopted. It rarely irritates, it removes odor, and often favors the healing of the sore.

Anthrarobin in the Treatment of Skin Diseases.—At a recent meeting of the Berlin Medical Society, BEHREND reported that he had used anthrarobin in cases where chrysarobin and pyrogallic acid were commonly used, with good results. Anthrarobin is a whitish-yellow powder, sparingly soluble in water, soluble in ten parts of glycerine at 212° F., in ten parts of cold, absolute alcohol and five parts of warm alcohol. It makes a good ointment with oil, and was used in ointments of ten and twenty per cent., in a tincture of ten per cent., and a glycerine solution of ten per cent. Anthrarobin is less irritating than chrysarobin, and can be used upon the face and eyelids. It colors the skin yellow, and a burning sensation follows its first application, which does not generally persist. It is well to precede the use of anthrarobin by the use of green soap, which will remove any obstacles to the action of the medicament. The effect of anthrarobin lasts longer than that of chrysarobin, and it is considerably cheaper. It is more potent than pyrogallic acid.—*Deutsche med. Wochenschrift*, March 8, 1888.

An Application for Sore Nipples.—SCARFF, in the *Maryland Medical Journal*, prescribes the following:

Balsam of Peru,
Tr. arnica . . . . . āā ʒss.
Expressed oil of almond,
Lime water . . . . . āā ℥ss.

Mix. Shake well, and apply to inflamed nipples with a camel's-hair brush, after cleansing them with borax and water.

An Urea Centre.—The *Lancet* writes that in the animal economy centralization is carried to its most extreme degree. It is certain, from Brown-Séquard's experiments, that there exists a centre irritation of which can suspend the chemistry of the body, making the blood in the veins flow red and reducing the temperature of the body. MM. Sée and Gley, experimenting on dogs with a view to producing glycosuria, caused azoturia by irritating the central end of the divided right vagus with a mixture of glycerine, water, and lycopodium injected into that part of the nerve after section.

Glycerite of Bichloride of Mercury for Antiseptic Use.—The *American Druggist* writes that there has been for a long time prepared for use in the public hospitals in New York, a glycerite of bichloride of mercury, containing one grain of the salt in every two minims. In addition, there is about a quarter of a grain of chloride of sodium in this quantity. When this solution is made, there is always a more or less decided formation of calomel (which is, however, greatly diminished by the use of the chloride of sodium). In order to make the final product as accurate in proportions as possible, the original solution is made somewhat stronger, and then allowed to become perfectly clear by standing, the clear liquid is poured off and, having added alcohol to the residuary portion, this is passed through a filter. The calomel retained by this is washed and dried. Making allowance for the amount of bichloride hereby lost from the original amount, the product is adjusted so that it will represent one grain of the mercurial salt in two minims. It is advisable to take about one-sixteenth part more of bichloride, at the start, than is theoretically required, so as to leave a sufficient margin for dilution. Such a solution, *slightly tinted with fuchsine*, would be very handy for carrying about with the field hospitals and medicine chests.

Purpura Hæmorrhagica Caused by Mental Shock.—DR. ED. DE SMET of Brussels, has recently had a case of a young woman of a highly nervous temperament who, in consequence of severe fright, experienced an eruption of purpura hæmorrhagica, though she had never had any eruption of the kind before. Some four months subsequently, however, in consequence of a fall, a similar eruption made its appearance. The treatment was directed mainly to the nervous system, which was evidently very much affected. Dr. de Smet refers to M. Lenoir of Lille, who has published cases of various skin diseases—eczema, psoriasis, herpes, pemphigus, and vitiligo—which have followed a mental shock.—*Lancet*, March 17, 1888.

The Treatment of Epithelioma by Chlorate of Potassium.—The Paris correspondent of the *British Medical Journal* of March 17, 1888, writes that DR. HYVERNAUD has treated sixty-three cases of epithelioma by local applications of chlorate of potash; of these, thirty-two were cured, fifteen were benefited, and sixteen cases were unrelieved. In the successful cases, the disease was situated on the skin of the face, nose, eyelids, cheeks, neck, in the lumbar region, on the back of the hand, and on the inner surface of the leg. Chlorate of potash fails as a rule when the mucous membrane is the seat of disease, although two cases of epithelioma of the lips and nostril were cured by it. It is especially useful when the affection is slow in its progress. A six per cent. solution slightly heated, or a fine powder of chlorate of potash, is applied to the wound, after it has been freed from scabs. The dressing is renewed once a day, or oftener. The effect of the solution is chiefly to heal the wound, while the powder slightly cauterizes it. The surface of the tumor or ulcer is thus destroyed, small eschars come away, and cicatrization ensues. In most cases, several weeks, or even months, are required to obtain complete cicatrization.

A Convenient Solution of Saccharin.—CHAMBERLIN, in the *Australian Journal of Pharmacy*, No. 26, 1888, reports the following convenient preparation of saccharin:

I have, as a sample, produced the following, and found it makes a pleasing reliable solution: Saccharin, gr. 80; potass. bicarb., gr. 40. Mix intimately in glass mortar. Add glycerine, ℥iss; aq. destil., ʒiss. Allow escape of carbonic acid, transfer to flask, and melt gently in water bath. This contains about gr. 1 in min. 20, sufficient for a cupful of milk.

Geranium Maculatum in Chronic Bronchitis.—The *Journal de Médecine* of March 11, 1888, gives the following as a useful prescription in chronic bronchitis and bronchorrhœa:

℞.—Tinct. nucis vom. . . . . ʒ i.
Tinct. sanguinariæ . . . . ʒ i.
Ext. fluid. geranii . . . . ʒ i½.
Syrup. simpl. . . . . ʒ ii¼.

Of this a drachm should be taken every four hours.

THE MEDICAL NEWS.

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OF MEDICAL SCIENCE.

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SATURDAY, APRIL 14, 1888.

ENGLISH AND AMERICAN CÆSAREAN RECORDS COMPARED.

THE late Dr. Thomas Radford, of Manchester, with very commendable zeal, prepared upon three several occasions—the last when he was eighty-seven years old—tabular records of the Cæsarean operations performed in the British Isles, from January 9, 1738, to May 20, 1879, aggregating 132 cases, with 109 deaths. We now add to this list 19, 7 of which he overlooked, making 151, with 122 deaths. So far, then, as known, there have been 29 women and 83 children saved by gastro-hysterotomy in England, Ireland, and Scotland, in a period of one hundred and forty-nine years. That our late friend and correspondent failed to note a number of cases is not to be wondered at, and these were not always fatal, although never published. The same process of personal solicitation will be found requisite in England for the obtaining of unreported cases as has been so effectual here in filling out our record. Such a work is very discouraging, because it brings to light such a picture of fatality; but it is only in this way that the misfortunes of the past can be studied, comprehended, and avoided. We know that Dr. Radford must have failed to secure records of many cases, which ought to be and might be still secured by a young and energetic worker. Mr. Henry Jackson, of Sheffield, and his son Arthur, both operated on the same woman at an interval of seven years, the second operation proving fatal; the case was never published. Mr. Lawson Tait operated twice (cases fatal) during Dr. Radford's day, but he failed to answer the published letters of the latter, asking for reports to be sent him. The late Mr. Samuel Smith operated at the Infirmary of Leeds, in 1863, and the late Mr. Samuel Hay, in 1865. Mr. Scattergood operated in Otley, in 1869; the women were all malacosteon subjects, and all died; but the children were saved. A search through the traditions of Glasgow, we are informed, will show a repetition of the work of Leeds; and a general investigation in many localities in England will accomplish what has been done in some of our individual States—as in Louisiana and Georgia; in the former, 10 out of 19 cases were never published, and in the latter, 7 out of 11.

We have now a record in the United States of 164 Cæsarean operations, of which 16 were Sänger cases. Of this list, 56 were never published, and would have been entirely lost to science but for the search made; many other cases were *communicated*, but were subsequently reported through the journals, or will yet be. 62 women and 63 children were saved; those born moribund, or that very soon died, are not counted in this record.

The Cæsarean operation is much more frequently performed in the United States now than in former years, the average per annum having increased from 3 to 6, there having been 24 in the last four years, which is not likely to be reduced, under the prospect of a diminishing fatality, by the adoption of the Sänger method. Great Britain does not yet appear to realize the capabilities of this valuable death-reducing improvement; possibly the three successes in order of Prof. W. T. Lusk, of New York, during the past year, who is one of the vice-presidents of the British Gynecological Society, may induce some of her members to make trial of it. Since May 20, 1879, when Dr. Radford's record closed, there have been published only 12 British Cæsarean operations, saving 4 women. We know that the number of cases has fallen off in latter years, but do not believe that this is even nearly all of them. From the date given to the present time, our own country numbers 40 cases. In the three years 1865, '66, '67, there were 20 British cases; in 1875, '76, '77, ten years later, there were, as far as known, 10; and in 1885, '86, '87, only 6.

ERYTHROPHLŒIN.

IN THE MEDICAL NEWS of March 10, 1888, we called attention to the experiments of Liebreich and

Egasse, Nevinny, and several other investigators on the action of haya or erythrophlœin, the new local anæsthetic, derived from an African plant. In the *Berliner klin. Wochenschrift* of February 27, 1888, is an additional communication by Liebreich, made to the Berliner med. Gesselschaft, and a discussion of his paper by Lewin. Again, in the *Therapeutische Monatshefte* for March, 1888, is a very full paper by Liebreich, while in the *Deutsche med. Wochenschrift* of March 8th is a discussion on a paper by Karewski, concerning the same drug.

Medical literature has been so burdened by reports of the action of the poison that it is at first difficult to reach a conclusion as to its true anæsthetic value. At present, however, its most enthusiastic upholders confess to its inferiority to cocaine, and there can be no doubt that it is possessed of far greater toxic powers than its sister anæsthetic. Enough clinical work has not been reported to enable us to point out any conditions in which it can succeed where cocaine has failed, and we doubt if any such properties will be discovered. The enthusiasm over its introduction is but another evidence of the yearning of humanity after anæsthetics which do not produce unconsciousness.

THE AGNEW JUBILEE.

ON Friday, April 6th, Professor D. Hayes Agnew passed the fiftieth anniversary of his entrance into the medical profession. This anniversary excited such widespread and kindly interest, that the word "jubilee" expresses most fitly the sentiment of his professional brethren on that occasion. This sentiment has found expression in the call for a jubilee meeting, to be held by his students, on April 24th, at the University of Pennsylvania, and reached its most felicitous demonstration April 6th, when the guest of the day was banqueted by a large assembly. Here, some of those who witnessed the work of his earlier years bore eloquent testimony, in prose and verse, to the value of his career, while the consensus of feeling and opinion exceeded the compliments usual on social occasions, and became heartily and affectionately congratulatory. And well they might be; for to have participated in the development of surgical science for fifty years with that intelligence which only anatomical study can bestow; to have seen the greatest enemy of the surgeon, septicæmia, in a large measure vanquished; to have shared actively by precept and example in this the greatest advance in the medicine of the century; and to have won the affectionate regard and high esteem of his guild, and of his fellow citizens is a career preëminently successful.

Such a career has influence, constant and potent; whether spoken or written, the utterances of such a mind, exemplified in personal sincerity and devotion to science, are the noblest plea that scientific medicine can make for the respect, the admiration, and affection of mankind. We join most heartily in the jubilee of this distinguished professor and surgeon, and wish him many years of enjoyment of the honors and usefulness so widely extended to him. We wish him, then, long life, for

> "He most lives
> Who thinks most, feels the noblest, acts the best."

THE army of medical doctors has been augmented by recent graduations, as follows:

Jefferson Medical College, Philadelphia	188
Louisville Medical College	95
College of Physicians and Surgeons, St. Louis	44
Western Pennsylvania Medical College. Pittsburg	34
Medico-Chirurgical College, Philadelphia	20

AT the meeting of the Paris Academy of Medicine on March 13, 1888, Professor William T. Lusk, of New York, was elected a corresponding member, receiving forty votes against ten for Sir William MacCormack and two for Victor Horsley, of London.

THE importation of smallpox from Italy has probably ceased for the time, at the port of New York. The Health Officer of that station has adopted more stringent measures and more thorough vaccination of immigrants coming from infected ports. The correspondence of that officer and the Health Boards of New York City and Brooklyn, bearing upon the alleged defective inspection of immigrant vessels, has been very pointed, and at times almost acrimonious. As a result, the less frequent introduction of smallpox may be expected.

DR. NETTER, of Paris, has found, in the saliva of three healthy persons, a pathogenic microbe capable of causing death in animals. It appears to be identical with the bacillus discovered by Friedländer in the lungs of patients having pneumonia, and known as the diplobacillus pneumoniæ of Weichselbaum. It is sometimes associated with the pneumococcus of Fränkel. Netter found this microbe rarely; he met with it only three times in 180 specimens taken from

105 persons. It was rapidly fatal when injected into the blood of guinea-pigs and mice, but had no effect upon rabbits. Man may share this immunity, or it may be transient, as proved by Thost, that Friedländer's microbe may exist innocuously in the nasal mucus of healthy persons. The author believes that pneumonia is always connected with the pneumococcus of Fränkel, and that the action of Friedländer's microbe has not yet been proved to be constant and pathogenic. In his opinion, it will be found to be rather an accidental concomitant, due to its presence in favorable regions of the air-passages, rather than a necessary agent in the production of pneumonia. Further research must be had in order to determine the action of this organism as a disease-germ.

The total reported mortality in 1887 in the State of New York was 96,453, of which 36.3 per cent. were under five years of age. The average death-rate stood at 17.5 per thousand. The population is here taken to be five and a half millions—a figure wanting in certitude, in view of the fact that there has been no census for seven years. The average rate of death in certain regularly reporting districts—almost wholly urban in character—was 23; while in other districts, chiefly rural, the rate ran at 18.4 per thousand. If, by reason of known defects of registration of deaths in some incompletely organized sections, the entire State be given this last-mentioned rate, the total deaths would have reached 101,200. Zymotic fatality was higher than during the three years past. Diarrhœal deaths were unusually numerous during the midsummer months, with a correspondingly great loss under five years. The percentage of mortality in that age in the month of July was as high as 52.7; in June and August it was 40 and 45.3, respectively.

Judge Ewell writes to the *Journal of the American Medical Association* that "there is absolutely no doubt whatever, that in publishing clinical lectures without the lecturer's consent, a legal wrong is committed, for which the courts give an adequate remedy," and he offers to furnish authorities for this statement.

The establishment of a "Stomatological Society," whose object is the scientific study of diseases of the mouth, the teeth, and adjacent parts, is announced at Paris. In view of the development which laryngology has attained, a pure "stomatologist" will be a curiosity, especially interesting to laryngologists and dental surgeons, who will be concerned in allotting territory to the new specialty.

English-speaking residents in Switzerland are exercised over the Swiss law, whose enforcement is threatened, which withholds license to practise from any but graduates of the Swiss medical colleges.

The tendency of those who minister to the soul to include the body in their sphere of action, has been repeatedly exemplified, notably in the "faith cure" superstition, at present prevalent in America. The physicians of Belgium have resented the usurpation of their functions by the clergy, and are endeavoring to repress what they consider unjustifiable interference in their field of work.

The Royal College of Physicians held its annual election for President on March 31st. Sir William Jenner declined to be again a candidate, having held the position for seven years. In the balloting, which followed the presidential address, the race was very close between Sir Andrew Clark and Dr. Quain, but the former was elected. On the first ballot Dr. Quain had the highest number of votes, but he fell short of a two-thirds majority, whereupon in the second ballot Sir Andrew ran ahead of him, having seventy-nine votes to Dr. Quain's seventy-one votes.

Under the title *Nouvelle iconographie de la Salpêtrière*, MM. Nicher, Tourette, and Londe announce the publication of a journal under Professor Charcot's direction, which will be a bimonthly review of the diseases of the nervous system.

The publication of an Italian edition of the *American System of Medicine* is announced, by Dr. Francesco Vaalardi, at Milan.

Albert G. Browning, M.D., of Providence, R. I., died April 6th, at the age of forty-nine years. The cause of his death was apoplexy, by which he was first prostrated March 8th.

Dr. Browning was born in Woodstock, Conn. After graduating from the Academy in that town, and teaching for a while in the schools thereabouts, he began the study of medicine under Dr. George H. Church, of Wickford, R. I., and subsequently graduated from the Yale Medical School. He practised medicine successfully in Providence and

vicinity about twenty-five years, and during the last half of that period gave much attention to the special uses and applications of electricity as a means of treating disease.

He was a cheerful, genial man, a ready speaker, and possessed a vast fund of witty anecdotes and stories. Politically he was a staunch Democrat. He had been a Fellow of the Rhode Island Medical Society since 1863. His wife and his aged mother are the only near relatives who survive him.

REVIEWS.

THE PRESCRIPTION. By OTTO A. WALL, M.D. St. Louis: Aug. Gast Bank Note and Lithograph Co.

THIS little book, while it contains quite a large amount of information as to the derivation of signs and symbols will, we believe, only prove useful to the student of pharmacy, or to the physician who has sufficient time to devote himself rather to the non-practical details of medicine than to those of daily life.

While many of the definitions are interesting because of their derivation, the book is marred by tautology to a very large extent, and lacks smoothness. The advice given is unsuited to the well-educated physician, and the book contains too much matter for the busy medical student, as is evident when the number of pages is considered, one hundred and seventy-seven. No book can ever avoid treading upon some one's toes, which tries to give advice to both the physician and druggist at one and the same time, yet this is the avowed object in his preface of the author of *The Prescription*. The book savors of placing the druggist on the same plane as the physician, which is a mistake, since, while the professions are to a certain extent linked together, they never can be identical.

SOCIETY PROCEEDINGS.

NEW YORK ACADEMY OF MEDICINE.

Stated Meeting, April 5, 1888.

THE PRESIDENT, A. JACOBI, M.D., IN THE CHAIR.

DRS. E. C. SEGUIN and ROBERT F. WEIR read a double paper entitled

A CONTRIBUTION TO THE DIAGNOSIS AND SURGERY OF CEREBRAL TUMORS,

which will appear in full in an early number of *The American Journal of the Medical Sciences*.

DR. W. W. KEEN, of Philadelphia, gave some details of the case briefly referred to in THE MEDICAL NEWS of December 24, 1887. The full report of the paper had not been published yet, he said, as he preferred to present it at the Washington Congress next September. At the age of three years the patient fell from a window, his head striking on a brick. A superficial wound was made, but no trouble was experienced from the injury until twenty years had elapsed, when epilepsy developed. This was associated with aphasia and paresis and paralysis of the right arm and leg. No history could be obtained of any "signal symptoms," on which Dr. Seguin had laid stress in the paper. Later there was complete recovery from the paresis, paralysis, and aphasia.

The operation was performed December 15, 1887. Instead of marking upon the skull with a colored pencil, the spot where the opening was to be made, as had been done by Dr. Weir in his case, a little piece of bone was gouged out at a point corresponding with a slight scar which was presumably left by the injury to the head received when the patient was three years old. The tumor removed measured nearly three inches in its long axis, and about two inches in its short axis. During the operation great trouble was experienced from hemorrhage, due in great part to the extreme friability of the vessels. The hemorrhage was controlled partly by hot water, partly by ligation, and partly by pressure. In tying a vessel it was found that if one end of the catgut was pulled at all more strongly than the other, the ligature would invariably cut through; so that absolute equality of traction was required in order to accomplish this successfully. The hot water employed was at a temperature of from 120° to 130° F.

The patient did perfectly well for two days after the operation. On the third day marked symptoms of pressure showed themselves, and this was ascertained to be due to the presence of a large clot, of greater size than the tumor itself. This was removed by careful washing, and all went well for ten days, when pressure symptoms were again observed. There was also severe diarrhœa and a temperature of 104½°. Under the circumstances, the presence of pus was suspected, and the wound was accordingly reopened. Afterward Dr. Keen regretted having done this, as it resulted in *hernia cerebri*, and he become convinced that the unfavorable symptoms noted were largely due to the diarrhœa. Later the patient had two or three other attacks of diarrhœa (less severe than the first), which were also accompanied by more or less rise of temperature. The hernia remained for a considerable time, and in order to facilitate the process of healing skin-grafts were transferred from the arm. The wound was dressed with bichloride gauze for eight weeks, but there was no evidence of its producing any toxic effect upon the system; the diarrhœa not being attributable to this cause. While the healing was going on the surface remained considerably elevated, but when the cicatrizing process had been completed it suddenly became concave, instead of convex, and has so continued ever since. Photographs were exhibited of the *hernia cerebri* and of the wound when healed.

Recurring to the operation, Dr. Keen said that he did not employ the spray, but made use of all the other ordinary antiseptic precautions. In speaking again of hemorrhage, he said that he regretted that previous to the operation he had not administered a dose of morphia, as recommended by Horsley, for the purpose of diminishing the blood-supply to the parts. In another case he should also be inclined to give ergot, and perhaps antipyrin, and he preferred to use cocaine locally if occasion required. Such agents, he thought, afforded a much better chance of controlling hemorrhage in this class of cases than the mechanical means commonly resorted to. He then spoke of the importance of completely shaving the head, for purposes of examination, of every patient in whose case there was any brain trouble

likely to call for operation, and said that in a case of epilepsy that had recently been referred to him, two scars were discovered when the scalp had been shaved. In regard to the question of drainage, in his case he had been compelled to keep this up for a much longer time than he desired. Such a course was rendered necessary, however, on account of the large clot and the discharge which followed it. According to Horsley, drainage should not be maintained for more than twenty-four hours, and he believed that this might suffice, except under special conditions, such as existed in the present instance.

NEWS ITEMS.

The Agnew Jubilee.—Over two hundred members of the medical profession of Philadelphia sat down to a banquet in the Foyer of the Academy of Music last Friday evening in honor of the fiftieth anniversary of the graduation in medicine of Dr. D. Hayes Agnew. Dr. Da Costa presided, and on his right sat Dr. Agnew the guest of the evening. There were also present a number of Dr. Agnew's professional friends from other cities, including Drs. Sayre, Weir, and Agnew, of New York, Tiffany, of Baltimore, Billings, Ford Thompson, and Yarrow, of Washington, Hunter McGuire, of Richmond, Weist, of Indiana, and Parkes, of Chicago.

Dr. J. M. Da Costa, after the cloth was removed, spoke as follows:

Fifty years ago, on this very day, there stood, with the honors of a University just received, a young man on the threshold of his life. His thoughts were the pleasant ones of the occasion; his aspirations had hardly taken shape; he was the popular comrade of the hundred and fifty-five whose real life, like his own, was to begin. Fifty years have passed, and their Agnew has become our Agnew of the many thousands of the American profession.

Honored Guest: In addressing you to-night I feel that I speak not simply for those who are gathered around you; nor for those in this Commonwealth whose interest will centre here; but for the whole profession who hold you in such esteem, and whose sympathetic thoughts, could they reach you, would come to you in messages of such good-will and affection as to overwhelm you with their warmth.

Your career has been, indeed, a remarkable one; and you must pardon me, and let the occasion be my excuse, if, in your presence, I allude to its success, and to the main causes of that success. Nor is it wholly unfitting in one to do so who has known you, and watched your progress with friendly interest, almost since you came to this city, to try your powers in a wider field. The training you brought with you as a rural practitioner of note was indeed valuable. Self-reliance, cool judgment under difficult circumstances, are not the least reward of a country physician's hard life. You enrolled yourself as a teacher of medicine in its most laborious branch, and fittingly took charge of a school which has been the nursery of famous anatomists and surgeons—where Godman's practical skill was displayed, and Joseph Pancoast laid the foundation of that intimate knowledge of the human frame which made him afterward so great a surgeon.

This Philadelphia School of Anatomy, in College Avenue, has, indeed, left its mark in the history of medicine. It has been to us what the Windmill Street School was to the London of William and of John Hunter, of Hewson, of Cruikshank, of Baillie, of Benjamin Brodie, of Charles Bell. Its rickety structure harbored not only anatomists—some of them your own pupils, who were to succeed you as celebrated teachers—but its dingy walls heard eloquent discourses on diverse branches from more than one of your future colleagues; in its garret, independent and fruitful researches on the textures of the body were pursued; in its cramped lower room, physiological experiments were carried on, which have made their deep impress on the science of our day.

For ten years working in this school of anatomy you lived laborious days and nights, and in its stern training your classes grew, until the narrow quarters would hold them no more, and you became the popular, admirable teacher you have proved yourself since, on a larger scale and on a different branch, as Professor of the Principles and Practice of Surgery in the famed University with which your reputation is forever identified. You learned to present facts plainly and impressively, to teach Nature's truths with Nature's simplicity, and without a deadly paralysis of words.

But in these ten years of unremitting work you did something more than teaching. You laid, by exact knowledge, by steadiness of purpose, and affability, the foundations of that large practice which you have since enjoyed, developing every day, more and more, into the trusted surgeon whose deft hand and cool judgment caused his advice to be generally sought. Every country shows in its professions the national traits. You certainly represent, as a surgeon, besides much skill, the American characteristic of resolute common-sense.

You have been tried in many a hard case. In none harder, than when your reputation caused you to be selected among the counsellors at the wounded couch of one for whose relief millions were anxiously watching. That in these trying times you bore yourself with the same calmness and dignity we know in you, every one in these millions recognized.

Your success as a surgeon of great repute must, indeed, have been gratifying to you. Not only for the opportunities it afforded you of doing so much active work in your profession; not only because it gave a personal value to your writings, especially to your opinions expressed in your elaborate work on *Surgery;* but because it enabled you to carry out a plan of action, of which I may not speak,—one which showed you to be possessed of the same high sense of honor for which Sir Walter Scott has received the unbounded admiration of mankind.

May you, dear sir, who have these many claims to distinction and esteem; may you on this, the fiftieth anniversary of entrance into a profession which you have graced by your industry, your sagacity, your skill, your character; may you accept the homage of those who are engaged with you in the same pursuit as a sign of widely felt regard and appreciation. May your vigorous frame preserve your power of doing good, of teaching truths, for many a long year. May there always remain with you the assurance that, as age gently lays its hand on you, the chilling finger of time will not lessen the respect, nor benumb the tenderness of feeling, with which old and young alike regard you.

DR. AGNEW, upon rising, was greated with enthusiastic cheers. He said

Mr. Chairman: When I glance over this table and see so many distinguished and representative men of my profession who have come to do honor to one of the humblest of their guild, I confess it is with a deep sense of embarrassment that I wish to respond to the complimentary sentiments embodied in your toast and in your address. I can say without affectation that had it been the pleasure of my friends, a less conspicuous demonstration of their goodwill than the present would have been more in consonance with my feelings and taste.

It is a great distinction for any man in whatsoever sphere of life he may move to win the confidence and esteem of his fellows, and it is scarcely necessary for me to say how profoundly I realize this mark of your respect. Any man who has lived long in this world, and has taken a thoughtful retrospect of his past life, must be forced to confess that the influences and forces which have conspired to mould his character, and to shape his destiny are most mysterious indeed. Plans constructed with infinite care have miscarried; fondly cherished hopes have been suddenly crushed with a shock, as when a precious vase is broken to fragments on the pavement; glowing anticipations just about to be realizations dissipated in a moment into thin viewless air like earth mists before the morning sun. One of Scotland's gifted but unfortunate sons has condensed these thoughts into the expressive couplet,

> "The best laid schemes, o' mice and man
> Gang oft aglee."

And the great bard of England, rather the bard of all peoples and all times, has crystallized the same sentiment in those expressive lines,

> "There is a divinity which shapes our end
> Rough hew them how we will."

And a greater than either has said it is not in man to direct his steps.

About thirty-five years ago I came to this city, a stranger. Though previously enjoying a large practice, in a wealthy and populous district, yet the field was never a satisfactory one. The horizon seemed too contracted, the prospects uninviting. Possessing naturally strong anatomical tastes, I was able to gratify these to only a moderate degree by dissections of the human body and the bodies of animals; yet the prejudice against studies of this nature was calculated to render their prosecution unpopular, not to say very embarrassing. The desire, therefore, to obtain a larger field for the gratification of the idol of my thought, induced me to make Philadelphia my home.

The scene of my early labors, as most of you know, was in College Avenue, in the Philadelphia School of Anatomy. This institution—not a chartered, but a private one—had both an ancient and honorable history. It was here that Godman and Webster and the elder Pancoast and Allen laid the foundation of their reputations. It was indeed a school of the prophets; a gymnasium in which were trained the great majority of the men who, as teachers and writers, have filled so large a place in the medical world. Among these names may be mentioned that of Gerhard, the pioneer of physical diagnosis in America; of Wallace, who became Professor of Obstetrics in the Jefferson Medical College; of Bridges, Professor of Chemistry in the Philadelphia School of Pharmacy; of Keating, elected Professor of Obstetrics in Jefferson College, but compelled to resign on account of ill health; of Henry H. Smith, Professor of Surgery in the University of Pennsylvania; of Frances Gurney Smith, Professor of Physiology in the same institution; of your honored Chairman, Professor of the Principles and Practice of Medicine in the Jefferson Medical College; of Penrose, Professor of Obstetrics in the University of Pennsylvania; of Brinton, one of the Professors of Surgery in the Jefferson Medical College; of Garretson, the author of a popular work on oral surgery; and of Keen, Professor of Surgery in the Women's Medical College of Philadelphia. It was here that Brown-Séquard delivered his lectures on operative physiology, and it was here that Mitchell conducted his classic experiments on snake poison, and on many physiological problems, which have placed his name alongside that of Ferrier, and given him a high place among the scientists of the present day. It was in this institution that for several years, summer and winter, night and day, rarely taking a vacation, I studied the mysteries of this wondrous frame, with a love and a zeal which never flagged, and drew around me—notwithstanding the hostility of the two great medical schools—from the small beginning of twelve medical students, the largest private classes, only limited by the capacity of the building, that had ever followed a private teacher in this country.

Those were happy days; Yes, I think the happiest days of my life. I was then master of my kingdom. Those old dingy rooms had more attractions for me than the frescoed and fretted walls of a palace, and those anatomical odors were as sweet as those of Araby, the blest. The curriculum of instruction consisted of lectures on descriptive anatomy, surgical anatomy, operative surgery, and dissections, and when I was induced to accept the demonstratorship of anatomy in the University of Pennsylvania it was with reluctance and regret that I left the Philadelphia School of Anatomy.

It is entirely natural that my thoughts should linger about College Avenue—for it was there that I formed those friendships which, like hooks of steel, have bound me to the men of every part of this country and not a few across the sea.

In 1854, a united effort was made to reopen the Philadelphia Hospital for medical teaching, the doors of which had been closed for several years. History furnishes innumerable instances in which the most momentous events have been determined by the most trivial incident. All these fade into insignificance when contrasted with that which closed the doors of this great clinic against the profession. The cackle of a few geese saved Rome, a presumptuous cock-roach barred the gates of the Blockley Hospital—and let me say here in parenthesis, that to Dr. Henry H. Smith and Dr. J. L. Ludlow more than all others combined, the profession and the country at large are indebted for the restoration of this important hospital to clinical instruction. By their unwearied and indefatigable perseverance, by their unceasing importunity, like the widow with the unjust judge, these men succeeded in revolutionizing the antagonistic and settled sentiment of the Board of Guardians, and gained this great conquest, and on the same day that the hospital committee made their favorable report, Dr. Smith and myself were

elected surgeons to the institution; and for twelve years, save for a short interregnum, when self-respect demanded the resignation of the Medical Board, I continued to discharge the duties of a clinical teacher of surgery in the hospital. In 1864, I was elected one of the surgeons to the Wills Hospital for Diseases of the Eye, fulfilling the duties of the place for a period of about six years. In 1865, I was honored by an appointment on the surgical staff of the Pennsylvania Hospital, and for about eleven years continued my service in that venerable institution until the inauguration of a policy which compelled me to resign. Let me say here that I have made it a rule of life never knowingly to violate my sense of duty—and with the help of God's grace, it has never cost me one moment's hesitation to turn my back on any proposition or place however tempting, the acceptance of which would compel me to surrender my conscientious conviction of right. In 1877, the Board of Managers, of its own motion and unsolicited, with a cordiality and unanimity which I can never forget, reëlected me to my former place on the surgical staff. In 1867, I was elected one of the surgeons to the Orthopædic Hospital, then situated on Ninth Street, and which has now grown to be one of the important institutions for the treatment of deformities in this country.

In 1870, the Trustees of the University elected me to the Chair of Clinical Surgery in that institution, and a year later to the Chair of Principles and Practice of Surgery, and on the completion of the University Hospital building I was appointed one of the clinical professors of surgery—why I should have been honored with so many appointments to places of dignity and influence, I know not. Other and abler men could have filled them. But this much I can say, I never schemed or planned for one of them. Any place in the republic of medicine was honorable enough to me. I knew there were golden apples in the garden of Hesperus, but I was too much in love with my own particular study to attempt the play of Hercules and the dragon.

My love for the profession was inborn, wrought into the very fibre of my mental organization, and inspired not by the honors at her command, but from the unspeakable satisfaction and pleasure of being able to use her resources for the benefit of our common humanity. How long I may be able to continue in this good service I know not. This I leave to the wisdom of Him who numbers the hairs of the head and notes the flight or the fall of a sparrow. When that supreme moment comes I shall be satisfied. I have had all that I could wish, more than I deserve, and now, gentlemen, this is a great honor which you have done me: to be the guest of such a company should satisfy the most exacting ambition, and let me say to each and all of my professional brethren here to-night, that I have no words which can fittingly express the profound obligations under which I am placed, to your generosity and kindness.

Dr. S. Weir Mitchell then read the following poem, which he had written for the occasion:

MINERVA MEDICA.

A golden wedding; fifty earnest years
This spring-tide day from that do sadly part,
When, 'mid a learned throng, one shy grave lad,
Half-conscious, won the Mistress of our Art.

Still at his side the tranquil goddess stood,
Unseen of men, and claimed the student boy,
Touched with her cool sweet lips his ruddy cheek,
And bade him follow her through grief and joy.

"Be mine," she whispered in his startled ear,
"Be mine to-day, as Paré once was mine;
Like Hunter mine, and all who nobly won
The fadeless honors of that shining line.

"Be mine," she said, "the calm of honest eyes,
The steadfast forehead, and the constant soul,
Mine the firm heart on simple duty bent,
And mine the manly gift of self-control.

"Not in my service is the harvest won
That gilds the child of barter and of trade;
That steady hand, that ever-pitying touch,
Not in my helping shall be thus repaid.

"But I will take you where the great have gone,
And I will set your feet in honor's ways;
Friends I will give, and length of crowded years,
And crown your manhood with a nation's praise.

"These will I give, and more; the poor man's home,
The anguished sufferer in the clutch of pain,
The camp, the field, the long, sad, waiting ward,
Watch for your kindly face, nor watch in vain;

"For, as the sculptor years shall chisel deep
The lines of pity 'neath the brow of thought,
Below your whitening hair the hurt shall read
How well you learned what I my best have taught."

The busy footsteps of your toiling stand
Upon the noisy century's sharp divide,
And at your side, to-night, I see her still,
The gracious woman, strong and tender-eyed.

O stately Mistress of our sacred Art,
Changeless and beautiful and wise and brave,
Full fifty years have gone since first your lips
To noblest uses pledged that forehead grave.

As round the board our merry glasses rang,
His golden-wedding chimes I heard to-night;
We know its offspring; in a hundred towns
His pupil children bless his living light.

What be the marriage-gifts that we can give?
What lacks he that on well-used years attends?
All that we have to give are his to-day—
Love, honor, and obedience, troops of friends.

Dr. Thomas Wistar also read an ode to Dr. Agnew.

A Protest against the Introduction of Water Gas into Philadelphia.—The College of Physicians of Philadelphia last week adopted a protest against the introduction of water gas into Philadelphia, based upon a report prepared by a special committee consisting of Drs. H. C. Wood, T. G. Wormley, J. W. Holland, H. Leffmann, I. Norris, Jr., and Homer Jayne.

The committee reported that the various analyses of water gas, as published, show that it contains from thirty to forty-five per cent., or even more, of carbonic oxide, while ordinary coal gas has in it from three to seven per cent. of the same compound. A comparison of the deleterious or poisonous substances yielded to the surrounding atmosphere during the combustion of an equal volume of each of the gases showed that the combustion of 100 cubic feet of coal gas would give rise to 55.84 cubic feet of carbonic oxide gas; whilst the combustion of an equal volume of water gas would generate 73.32 cubic feet of carbonic oxide. That is, the combustion of

a given volume of water gas would produce over thirty per cent. more carbonic oxide than the combustion of an equal volume of coal gas, and would, therefore, contaminate about one-third more of the air of a room.

In regard to the relative poisonous properties of the two gases before they are burnt, the report says, it depends upon the quantity of carbonic oxide in them, the other ingredients being comparatively harmless. When quantities of either gas get into a room a certain proportion escapes through the windows, doors, and other apertures, so that it does not follow that a gas containing twice as much poison as another will be doubly as fatal. According to most authorities, one-half of one per cent. of carbonic oxide in the atmosphere of a room is sufficient to cause death. The presence of one per cent. of coal gas would necessitate the presence of six-hundredths of one per cent. of carbonic oxide, which would not be sufficient to produce immediate bad results. On the other hand, one per cent. of water gas would be equivalent to from thirty to forty-hundredths of one per cent. of carbonic oxide. It is plain that three per cent. of coal gas in the atmosphere would do no serious injury, whilst but little over one per cent. of the water gas would be required to take life.

The introduction of water gas into cities has, so far as the committee has been able to get at the facts, always been accompanied by an extraordinary increase in the number of deaths from gas poisoning. In New York City in the seven years terminating January 1, 1888, there were 177 deaths from water gas and nine deaths from coal gas, whilst the number of deaths in January of this year in New York City from water gas is said to have equalled all the deaths from coal gas in Boston in the last fifty-five years. In the five years ending January 1, 1885, there were in the city of Baltimore forty-five deaths from water gas. In the year 1887, following the introduction of water gas into San Francisco, the number of deaths was twelve. Eleven deaths were reported in the New York newspapers as having occurred in Chicago in 1886 and 1887. Of the exact number that really did occur in Chicago the committee has no accurate information.

The committee has been very much struck by the fact that whenever they have met with the report of deaths occurring in a small city from the inhalation of gas, upon looking into the list of cities, supplied with water gas, they have invariably found that the city in which the death took place was upon the list. Within the week an occurrence in Reading, Pa., points out a very important danger. It is generally thought that these deaths are the result of carelessness in leaving the gas turned on, although extinguished. It would appear, however, that owing to the variations in pressure, and probably also to the variation in the mixture of the constituents of the water gas or to other cause, when the gas is left burning low at night it is entirely possible for the light to go out, and afterward sufficient gas to escape into the room to cause death. Thus on March 30th, in the Keystone House, in Reading, two gentlemen were found dead in their beds from water-gas poisoning, and, according to the testimony at the Coroner's inquest, the gas had been left by them lighted.

According to Mayor Fitler's annual report, over 12 per cent. of the yearly product of the Philadelphia Gas Works is lost in leakage, equal to 440,340,319 cubic feet annually. While it is not possible to form a definite opinion of the extent of ill-health caused by breathing noxious gases that escape in this manner, the committee believe it a subject for urgent consideration in selecting a gas for use in a large city like this.

Dangers that already exist from this source would be alarmingly multiplied by the substitution of water gas for coal gas. In Troy, N. Y., during the winter of 1886–87 fifteen persons were overcome by the escape of water gas from a leak in the soil, and required medical attendance to revive them. The inhabitants of an entire row of houses in the same city were similarly overcome, and in the cases which terminated fatally death was attributed to carbonic oxide poisoning.

It has been ascertained from Mayor Fitler that sufficient water gas would be introduced into the gasometers in this city to constitute one-third of the entire bulk used, and that the manufacturers who will bid for the contract to supply the water product admit that it will contain 30 per cent. of carbonic oxide. After it is mixed with the coal gas, the substance which is supplied for illuminating purposes will contain 13½ per cent. of carbonic oxide. There is no limit of safety in the percentage of carbonic oxide, the report says, and the line must be drawn arbitrarily. The committee believe that 10 per cent. ought to be the maximum in any gas intended for use by the public as an illuminant. In Massachusetts a protest against the use of water gas containing more than 10 per cent. of the deadly vapor was sent to the Legislature with the signatures of all the leading physicians in the State, including the professors of Harvard College, and the medical examiners and health officers of the various counties and towns.

In conclusion, the committee recommended the adoption of the following, which was endorsed by the College of Physicians:

Whereas, the College of Physicians of Philadelphia has learned that it is proposed to furnish the citizens of Philadelphia with illuminating gas consisting of two parts of coal gas and one part of water gas, a mixture which would probably contain at least about 15 per cent. of carbonic oxide; and

Whereas, in the opinion of this College, the use of any illuminating gas which contains more than 10 per cent. of carbonic oxide would be attended with very grave risks to the health and lives of the citizens of Philadelphia; therefore, be it

Resolved, That the Mayor of Philadelphia is hereby petitioned that if, in his opinion, it be necessary to use water gas in connection with illuminating gas, that great care be taken to render it certain that the mixture shall at no time contain more than 10 per cent. of carbonic oxide.

New York Neurological Society.—At the meeting held April 3d, the following officers were elected:

President.—George W. Jacoby, M.D.

Vice-Presidents.—C. L. Dana, M.D., and M. A. Starr, M.D.

Recording Secretary.—B. Sachs, M.D.

Corresponding Secretary,—E. D. Fisher, M.D.

Treasurer.—G. M. Hammond, M.D.

Councillors.—I. Weber, M.D.; E. C. Seguin, M.D.; J. C. Shaw, M.D.; W. R. Birdsall, M.D.; E. C. Harwood, M.D.